THE BRITISH JOURNAL

OF

OPHTHALMOLOGY

NOVEMBER, 1931

COMMUNICATIONS

A CONTRIBUTION TO THE PATHOLOGY OF PAPILLOEDEMA

BY

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Introduction

IT is generally held that in papilloedema the swelling does not affect the whole disc at once, but starts at one or other part of it. Some, following Horsley,[1] state that the swelling is first seen at the upper and inner quadrant, while others believe that it appears first at the upper or lower border of the disc. It is universally agreed, and Gowers[2] was especially insistent upon this point, that the inner (nasal) edge of the disc is affected before the outer (temporal). No satisfactory explanation of this special site of commencement of papilloedema has been advanced. Another point which appeared to us to call for explanation is the reason why in papilloedema, considering the theories of its causation most widely held, practically speaking, only the disc and its immediate neighbourhood should be considered to be affected.

The theory of the method of production of papilloedema most widely adopted in this country is that based on the work of Deyl,[3] of Dupuy-Dutemps[4] and of Paton and Holmes,[5] and may be stated shortly as follows. The increased intracranial pressure is transmitted to the subarachnoid space round the optic nerve since this

space is continuous with the intracranial subarachnoid space (Schwalbe[6]). As they cross the subarachnoid space the central retinal vein and its accompanying lymphatic vessels are compressed with resultant dilatation of the retinal veins, oedema of the disc, and later, exudates and haemorrhages.

But the central retinal vein is an end vein, apart from a very slight anastomosis at the entrance of the optic nerve which is not to any extent comparable with the arterial circle of Haller, and we are therefore led to inquire why the whole of the retina drained by the

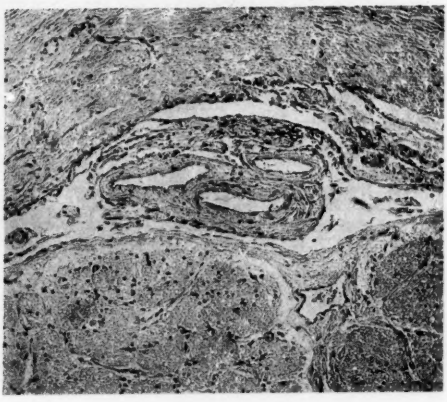

FIG. 1. × 105*

Transverse section of human optic nerve to show the central retinal vessels in the peri-optic subarachnoid space.

central vein should not be equally affected. A venous anastomosis of any considerable extent at the nerve head would evidently tend to carry away excess of fluid rather than hinder its passage. The same criticism would apply to the theory that attributes papilloedema to the raised intracranial pressure damming back the lymph stream which normally is said to flow from the eye along the optic nerve towards the chiasma. (Parinaud,[7] Ulrich,[8] Sourdille,[9] Kampherstein,[10] Rochon-Duvigneaud,[11] Liebrecht,[12] Behr[13]).

Schieck[14] is of the opinion that the subarachnoid space around the optic nerve communicates directly with lymphatic spaces around the central retinal vessels so that any increase in intracranial pressure would result in the *cerebrospinal fluid* being forced into the optic nerve along the central retinal vessels causing all the phenomena of papilloedema. Thus the swelling of the nerve head, produced by a distension of the perivascular lymphatic sheaths, will first be seen

* All microphotographs have been reduced one-third in reproduction.

at the bottom of the optic cup, pushing forward the internal limiting membrane, and subsequently the swelling will be visible at the places where the large vessels cross the edge of the disc. Schieck's theory of the method of production of papilloedema would thus present an explanation for the onset of papilloedema at a certain definite part of the nerve head. But the direct continuity of the subarachnoid space with the perivascular lymphatics about the central retinal vessels has not been definitely established, and the statement that the lymphatics of the retina open into the subarachnoid space requires further consideration. Indeed it is doubtful if true lymphatic vessels exist in the retina (which is part of the central nervous system). Schmidt-Rimpler[15] first suggested that it was actually the cerebrospinal fluid which got into the nerve and caused the swelling of the disc. Papilloedema would then be a sort of (rather inefficient) safety valve for the escape of cerebrospinal fluid in cases of increased intracranial pressure. Schmidt-Rimpler injected Berlin blue into the intracranial subarachnoid space of rabbits, dogs and calves, and subsequently found the dye in the lamina cribrosa. Leber[16] and others repeated these experiments but could not find the dye in the nerve and concluded that in Schmidt-Rimpler's experiments the dye must have got into a blood vessel. (See Fig 5, cat). Furthermore, Schwalbe,[6] in his classical experiments, found that it was not possible to inject the retinal lymphatic vessels from the subarachnoid space, whereas the dye would pass into these vessels if the syringe needle were placed ***under the pia mater***. Schieck himself repeatedly injected dyes into the subarachnoid space of human eyes (removed post-mortem with a length of optic nerve attached), and found that when a ***non-diffusible*** dye was used the particles of dye did not leave the subarachnoid space. This worker believed, however, that the fluid part of the dye passes into the nerve, as evidenced by the oedema of the spaces around the vessels similar to that which he noted in cases of papilloedema. But oedema has always been the bugbear of the histo-pathologist, and it is well known that oedema may not be evident in a microscopic section of an organ where oedema has been observed during life and, ***vice-versa***, the microscopic appearance of oedema in a normal tissue may be produced in the process of its histological preparation (imbedding oedema). In his later papers Schieck admits the possibility of this interpretation of his results, though in one case[17] of a freshly excised human eye he claims to have got the dye (Prussian blue—turpentine mixture of Baum[18]) into the retinal lymphatics by injecting it into the subarachnoid space around the attached length of optic nerve. So far as we are aware this is the only case on record in which the retinal lymphatic vessels have been injected directly from the peri-optic subarachnoid space. If there were a direct communication between the subarachnoid

space and the retinal lymphatics it should be possible to inject a coloured dye (diffusible or non-diffusible) by this route into the nerve and eventually produce a coloured papilloedema the evolution of which could be observed with the ophthalmoscope. With this main object in view the present writers performed a series of injection experiments on living animals. During the course of the present work a number of interesting facts emerged which are recorded below.

Personal Observations

The animals used were dogs, cats and rabbits. They were anaesthetised with a chloroform-ether mixture and the anaesthesia maintained by means of urethane (one gram. per kilo. body weight) injected intra-peritoneally. A few drops of a one per-cent. solution of atropine were instilled into each conjunctival sac to dilate the pupils.

In our earlier experiments we used a needle and syringe and, while one of us injected the dye (Indian ink) into the subarachnoid space either through the posterior occipito-atlantal membrane or through the cranial dura mater after trephining the skull, the other examined the fundus oculi with an electric ophthalmoscope. In these experiments it was found that the dye always passed into the subarachnoid space around the optic nerve even if only light pressure were made on the syringe. No dye, however, appeared in the eye. These early experiments were made on three rabbits, five cats and one dog. This method of injection was considered unsatisfactory as it was not possible to judge the pressure under which the dye was injected and, further, the dye tended to escape around the entrance of the needle.

In a second series of experiments we excised the eyes of living animals including a length of optic nerve with its surrounding meningeal sheaths, and injected the dye (Indian ink) directly into the subarachnoid space by means of a needle and syringe. This series comprised four rabbits, two cats and one dog. The dye (non-diffusible) did not pass into the eye as determined both by ophthalmoscopic and by microscopic examination. When the dye was forced with great pressure into the subarachnoid space it burst through the side of the disc into the vitreous but no dye was found in the optic nerve on subsequent microscopic examination.

In our later work we adopted a method of injecting a dye into the cranial subarachnoid space which is a modification of that employed by von Schultén,[19] and later by Cushing and Bordley,[20] in their attempts to produce papilloedema experimentally. A mid-line incision was made through the scalp down to the bone. A flap containing the temporal muscle was reflected to one or other side and a trephine opening, 10 mm. in diameter, was made over the

parietal eminence. The exposed circle of dura was excised (including the subdural layer of arachnoid) and a flow of cerebrospinal fluid resulted. Into the trephine hole a tightly fitting metal cannula was screwed, and this was connected by means of rubber tubing and a Y-piece glass connection, to a mercury manometer and to a Woulfe bottle containing the dye. The Woulfe bottle was connected to a large bottle which in its turn was connected to the water tap.

At the beginning of the experiment the parts of the apparatus between the Woulfe bottle and the metal cannula were filled with the warmed injection fluid. Then by turning on the water tap the pressure in the apparatus could be raised to any desired amount and the dye forced into the cranial subarachnoid space. Further, through the cork of the large bottle passed a short piece of glass tubing to the upper end of which a short length of rubber tubing was attached. The rubber tubing was closed by a clip, removal of which immediately lowered the pressure in the whole apparatus. Thus, if the pressure were raised too high and the animal became distressed, removal of the clip was followed by drop in pressure and rapid revival of the animal. It was found that, unless the veins in the neck were tied, there was a variable and often rapid loss of injection fluid from the apparatus on raising the pressure, undoubtedly due to absorption from the subarachnoid space into the venous blood stream; but by regulating the flow from the water

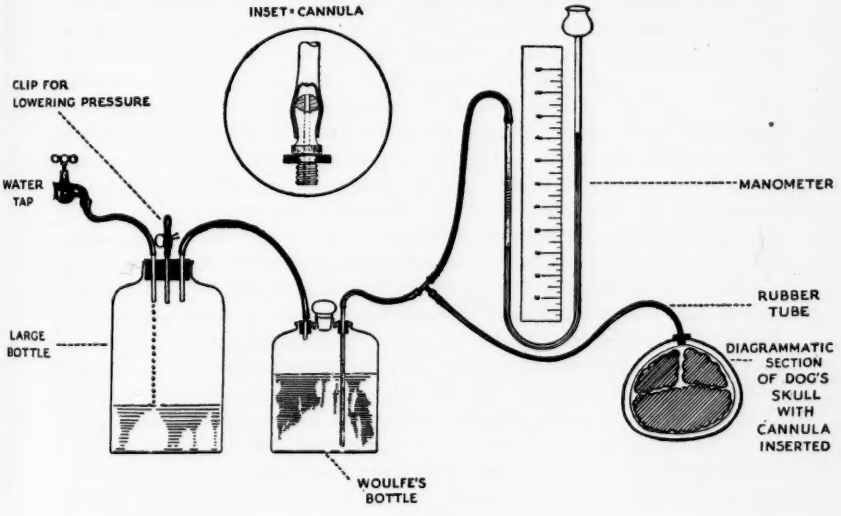

FIG. 2.

Diagram of INJECTION APPARATUS.

tap the pressure could be raised or kept constant as desired. Cushing and Bordley controlled the pressure at which they injected the fluids by the gravity method, namely, by raising and lowering a flask containing the dye. The experiments of this nature that we conducted comprised those on one rabbit, two cats and twenty-one dogs. We employed as injection material, substances of two types:—

(1) Those which were in suspension and were unable to pass through an animal membrane (*i.e.*, were non-dialysable); and

(2) Those which were in solution and were able to pass through an animal membrane (*i.e.*, were dialysable).

Substances in the first class we refer to as ***non-diffusible*** while those in the second class we label ***diffusible***. The non-diffusible substances we used were Prussian blue and Indian ink and the diffusible substances were methylene blue, eosin and Wasserblau.

Since the normal cerebrospinal fluid for all practical purposes is an extremely dilute aqueous solution (in fact, under normal conditions the nearest approach to water in the body), to prove that it is possible for cerebrospinal fluid to be forced into the retina and optic nerve if the intracranial pressure is raised, it seems only necessary to demonstrate that a diffusible dye can be forced in the living animal from the cranial subarachnoid space into the optic nerve. But there is a great difficulty in establishing this, because the diffusible dye soon diffuses into the blood stream and appears in the vessels of the eye. Thus, when we injected methylene blue into the cranial subarachnoid space the dye soon appeared in the retinal vessels and their change to a bright blue colour formed a striking picture when viewed with the ophthalmoscope. However, if the dye actually passed from the subarachnoid space into the optic nerve and retina, it ought eventually to produce a coloured papilloedema, if the hypothesis of Schieck and Schmidt-Rimpler is to be sustained. But this we failed to produce even though we raised the pressure in one case to 350 mm. of mercury (a pressure evidently incompatible with life). Another unsatisfactory feature concerning the employment of diffusible dyes for injection is that subsequent microscopic examination teaches us very little regarding the distribution of the dye during life. In some of our experiments we employed the method Weed[21] used in his studies of the cerebrospinal fluid. The injection fluid is a mixture of potassium ferrocyanide and iron ammonium citrate, in equal parts, made up to a strength of one per cent. in distilled water. After about three hours injection, the eye is removed and placed immediately into a solution of ten per cent. of formalin to which hydrochloric acid has been added to a concentration of five per cent. Only in two cases (one dog and one rabbit) in which, however, the pressure had been raised to 270 mm. of mercury, were any Prussian blue particles found in the optic nerve and (a very small amount) in the disc. Such a pressure had,

however, killed the animal so that it is probable that in these cases we are dealing with post-mortem diffusion. A negative result in these cases would have been evidence of the impossibility of forcing even a soluble dye into the nerve, whereas the positive result obtained is not conclusive evidence of the presence of direct communication between the subarachnoid space and the interior of the nerve (lymphatic vessels or tissue spaces). Certainly in those cases where the pressure was kept at a level compatible with life (70 mm. of mercury) no Prussian blue particles were observed, on subsequent microscopic examination, either in the nerve or in the disc.

The ***non-diffusible*** dye that we employed most frequently was Prussian blue, prepared immediately before the experiment by mixing equivalent amounts of potassium ferrocyanide and ferric chloride solutions, in this manner obtaining the finest particles of Prussian blue in suspension for injection. Using this injection fluid we found that, at pressures compatible with life (60 to 70 mm. of mercury), no blue colour appeared in the disc when viewed with the ophthalmoscope, nor were any Prussian blue particles seen on microscopic examination either in the optic nerve or in the disc. In some cases we raised the pressure to 270 mm. of mercury, and in one of these the particles were seen with the naked eye circulating in the episcleral blood vessels just beyond the edge of the cornea. In most of these cases the Prussian blue particles were found, on microscopic examination, in the choroidal vessels but not in the supra-choroidal space. In one case only (cat) a very minute quantity of the Prussian blue particles was seen, on microscopic study, in the optic disc, but they were contained in definite small vessels (apparently venules). (Figs. 5 and 6.) The presence of the particles of Prussian blue in the vessels is no doubt to be explained by the probability that the greatly increased intracranial pressure, in distending the subarachnoid space around the optic nerve, produces a rupture of some of the vascular trabeculae crossing this space. The Prussian blue particles would then pass into the torn vessels and thence into the choroidal vessels. Adamkiewicz[22] similarly found the dye in the choroidal veins in the rabbit, and concluded that distension of the choroidal veins was an accompaniment in clinical cases of raised intracranial pressure. However, it must be noted that the pressure employed in our experiments in those cases in which the particles of Prussian blue appeared in the veins was incompatible with life.

The effects of injecting fluids under pressure into the intracranial subarachnoid space may be considered under the following headings:—

1. Proptosis. 2. "Chemosis." 3. Dripping of fluid from the nose. 4. ? Papilloedema.

Proptosis.—Bilateral proptosis was observed in all the experiments in which a dye was injected into the intracranial subarachnoid space. It was due to oedema of the orbital tissues, as was shown by dividing the conjunctiva and ocular muscles and drawing the eyeball forwards thus exposing the retro-ocular orbital tissues (Fig. 7). In those experiments in which the pressure was kept about 70 mm. of mercury, the oedema fluid was found to be coloured when a diffusible dye was used and colourless with non-diffusible

FIG. 7.

Dog's eye drawn forward by dividing conjunctiva and ocular muscles to show retro-ocular oedema.

dyes. If the pressure was greatly raised and continued for some time after the death of the animal, even in those cases where a non-diffusible dye was used, particles of the dye were found in the oedema fluid. (Fig. 3.) Levinsohn,[23] who also noted this oedema of the orbital tissues, was of the opinion that the fluid passed from the subarachnoid space around the optic nerve through the dura mater at several places in the dog, whereas in the ape the place of exit was limited to the site where the central vessels pierce the dura. This experimentally induced oedema of the retro-ocular orbital tissues undoubtedly explains the proptosis which is not infrequently observed in clinical cases of cerebral tumour.

"*Chemosis.*"—In many of our experiments there appeared what was apparently a marked swelling of the conjunctiva, the swelling

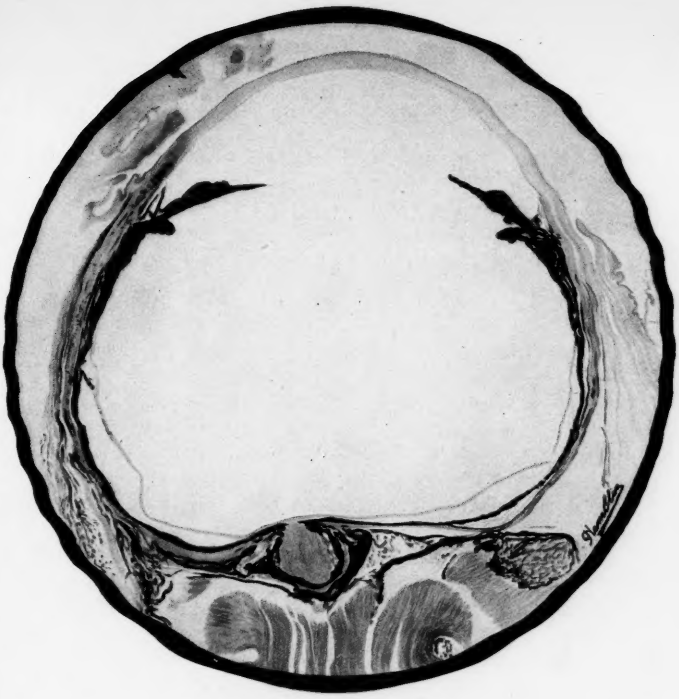

FIG. 3.

Horizontal section of dog's eye to show the effect of injecting potassium ferrocyanide into the cranial subarachnoid space at high pressure. Stained eosin.

The dye is in the subarachnoid space, outside the dura, in the orbital tissues, in the choroidal and scleral vessels and passes forward under Tenon's capsule to the conjunctiva.

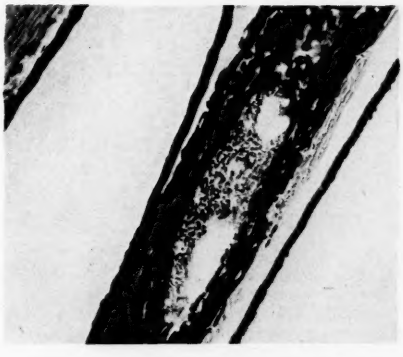

FIG. 4. × 200

High power view of choroidal vessel containing the dye. Note that the perichoroidal space is free from dye.

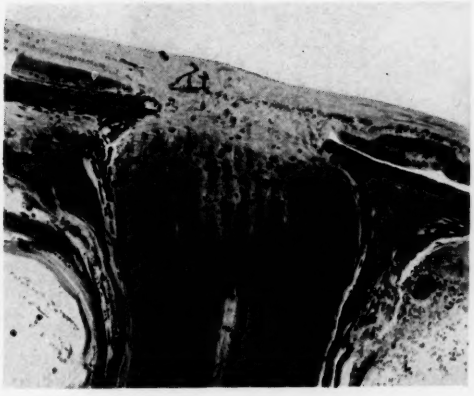

FIG. 5. × 55

Longitudinal section of the optic nerve head of a cat injected as Fig. 3. The dye is in the subarachnoid space and in some vessels (? venules) in the disc.

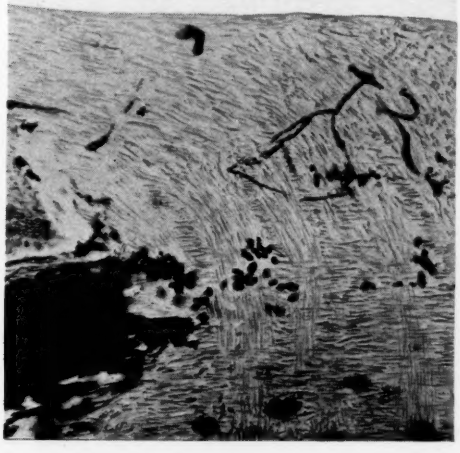

FIG. 6. × 300

High power specimen of injected vessels (? venules) in the disc seen in Fig. 5.

appearing in some cases as a roll between the eyelids (Fig. 8). On dividing the conjunctiva, however, it became evident that the fluid was not in the conjunctiva but under Tenon's capsule. When diffusible dyes were employed the fluid beneath Tenon's capsule was coloured in those cases where the pressure had been kept about 60 to 70 mm. of mercury. On the other hand, when non-diffusible dyes were used the fluid was colourless. In a few cases in which

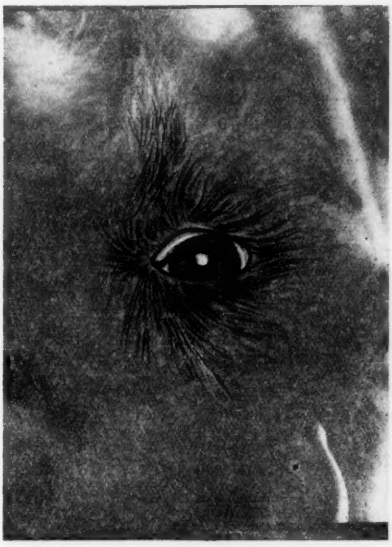

FIG. 8.

Dog's eye to show "chemosis."

the pressure had been greatly increased (270 mm.) and maintained for some time (about two hours) after death, Prussian blue granules were found in the fluid under Tenon's capsule. (Fig. 3.)

Dripping of Fluid from the Nose.—A constant sequel of injecting fluids under pressure into the cranial subarachnoid space is an escape of fluid from the nose. In those experiments in which diffusible dyes were injected, the fluid dripping from the nose was coloured, and first appeared when the pressure employed was in the vicinity of 40 mm. of mercury. When Prussian blue was employed the fluid dripping from the nose was colourless at pressures compatible with life. In one case only, in which Prussian blue was injected at a pressure of 370 mm. of mercury for two hours after

death, the fluid dripping from the nose was blue in colour. In three experiments (dogs) potassium ferrocyanide, made up in a one per cent. solution in distilled water, was used as injection fluid, and the fluid which dripped from the nose when the pressure approached 40 mm. of mercury was allowed to drop on a filter paper moistened with ferric chloride solution. The resultant Prussian blue reaction showed that at this pressure the potassium ferrocyanide solution was passing from the cranial subarachnoid space into the nasal fossae. At the conclusion of these three experiments the heads of the dogs were cut (post mortem) in median sagittal section, the cut surfaces dried with blotting paper and subsequently touched at various points with a glass rod moistened with a solution of ferric chloride. An intense Prussian blue reaction was obtained all over the cut surfaces, including mucous membrane and muscles.

These experiments demonstrate that, whereas diffusible dyes pass readily from the subarachnoid space into the nasal fossae at pressures compatible with life, particulate matter does not pass under these circumstances. This is evidence against the presence of direct communications betwen the cranial subarachnoid space and the nasal fossae. In this connection the work of Le Gros Clark[24] is of interest. This observer found that a solution of potassium ferrocyanide and iron ammonium citrate, dropped into the nasal cavities of living rabbits, reaches the cranial subarachnoid space by way of the perineural sheaths of the olfactory nerves, and he postulates the existence of a current running centripetally in these sheaths under normal conditions. Clark states that the evidence indicates that the perineural spaces are directly continuous above with the cranial subarachnoid space, though it must be remembered that he was dealing with diffusible substances and not with particulate matter.

? Papilloedema.—Before we could assess the value of any changes in the optic nerve head that might be produced by our experimental injections, it was necessary to determine the normal appearance of the "papilla optica" in these animals* and compare it with that of the human nerve head. The descriptions of the latter in our classical text-books differ to a marked extent, and it is therefore appropriate to give here some of the views expressed with regard to the appearance of the human nerve head.

The Human Nerve Head.—Briggs[25] in his "Ophthalmographia" (1676), was the first to describe the human nerve head and called it the ***papilla*** optica. This description was undoubtedly based on the examination of post-mortem material, in which a swelling of

* Up to the present it has only been possible to make a detailed study of the nerve head in the dog (and man).

the disc is normally present. Merkel[26] in the first edition of Graefe-Saemisch (1874), states: "Papilla nervi optici is a name which to-day has only a conventional meaning, as it has long been shown that during life there is no elevation of the nerve head above the level of the retina which justifies it." Fuchs,[27] in his text-book of Ophthalmology (1917) says: "The name 'Papilla' was selected by the older authors under the *erroneous* impression that the head of the nerve represented a projection into the interior of the eye . . . In the normal state it is perfectly flat so as to lie in the same plane as the retina." Testut[28] (1923) states that "the region of the papilla does not project as one would suppose from the name 'papilla.' Normally it is flat and situated exactly in the same plane as the retina of which it forms a part. But custom has made this name (papilla) sacred and for a long time yet we shall have to accept it (*s'incliner devant elle*)." Most modern text-books of anatomy and of physiology describe the nerve head as a projection and call it a papilla without qualification. It would seem, therefore, that the nerve head described as a papilla in 1676, became flat somewhere about 1870, and has now become a projection again! The present writers are of the opinion that this varied description of the nerve head is due mainly to the time and the method of examination of the disc. On ophthalmoscopic examination of the normal eye in the living subject, no swelling of the disc is seen and the vessels pass across the edge of the disc *without loss of their light reflex*. Also, if a freshly enucleated normal human eye be divided equatorially and the disc be examined by direct vision, no swelling of the disc is seen. If, however, the eye be first fixed in Zenker-acetic fluid for about 24 hours, sufficient swelling of the disc is produced to be just visible to the naked eye. It is to be noted, however, that the outer (temporal) side of the disc does not show any appreciable swelling. Microscopically, also, after the eye has been fixed as above described, the nerve fibres are seen to bulge in slightly towards the vitreous, not, however, sufficiently to deserve the name "Papilla" (*cf.* Salzmann[29]), and here again the fibres on the temporal side of the disc are seen to be on the same level as the neighbouring retina.

The Canine Nerve Head.—After fixation in Zenker-acetic solution we noted that the nerve head in the dog appears distinctly swollen to the naked eye. The swelling is greater than in the human, and differs from that in the human in that the projection is equally prominent on all sides of the disc, the temporal side being quite as prominent as the remainder. The absence of a thinner temporal side of the disc is undoubtedly to be correlated with the absence of a macula in the dog. It is essential to bear in mind the microscopic appearance of the normal canine disc after fixation in Zenker-acetic for the proper appreciation of the condition of the nerve head in

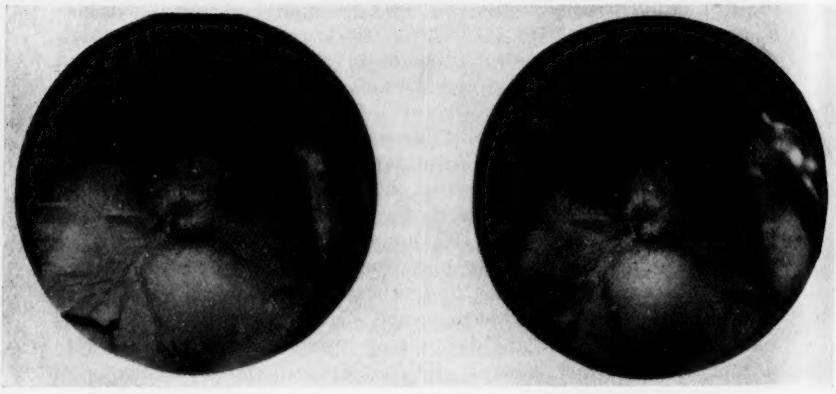

FIG. 9.

Normal dog's eye fixed within half an hour after death in Zenker-acetic to show the "swelling" of the disc that occurs.

those cases in which the intracranial pressure has been increased. The fluid used for histological fixation has also to be borne in mind as the appearance of the disc varies with the fixative employed, for example, Fig. 12 shows a section through the disc of a dog whose intracranial pressure had been raised to 70 mm. of mercury for two hours, by injecting Weed's fluid, the eye then being removed and fixed in 10 per cent. formalin and 5 per cent. HCl. Despite the increased intracranial pressure the disc does not project towards the vitreous like that of a normal dog whose eye has been fixed in

FIG. 10.

Longitudinal section of nerve head from normal dog's eye removed during life and fixed immediately in Zenker acetic. *Note the "swelling" of the nerve head.*

Zenker-acetic (Fig. 10). It is evident, therefore, how important is the method of fixation in judging the presence or absence of swelling of the disc from a microscopic section. If the eyeball of a living dog be removed, divided equatorially, and the vitreous taken

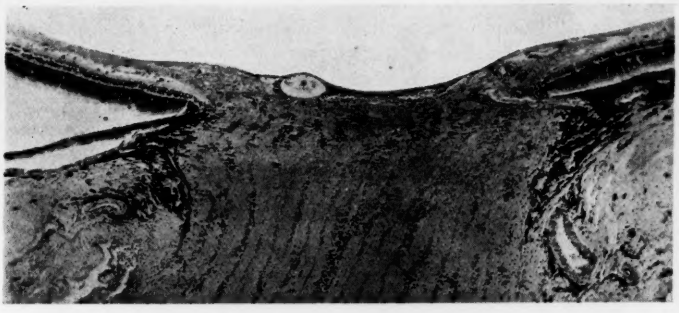

FIG. 11. × 55

As Fig. 10 but fixed in 10 per cent. formalin. There is no "swelling."

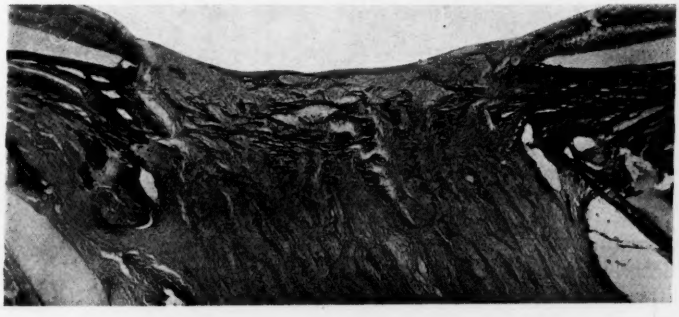

FIG. 12.

Longitudinal section of nerve head of dog whose intracranial subarachnoid space had been injected under high pressure. (250 mm. Hg) with Weed's fluid and then fixed in 10 per cent. of formalin + 5 per cent. HCl. Compare Figs. 10, 11 and 12.

out, no swelling of the disc can be seen either with the naked eye, a loupe, or with the slit lamp, whether the disc be looked at directly or after examining a section passing through it. With the slit lamp, the edge of the disc is seen to be markedly irregular with short whitish processes extending centrifugally from its entire circumference, giving the frayed appearance of opaque nerve fibres. The

blood in the retinal vessels is also seen by this method to move with gravity long after the eye has been removed. This, as Salzmann[29] pointed out, is important to bear in mind in interpreting congestion in a microscopic section of the eye. Thus with a slowly acting fixative the lowermost part of the eye will show vascular engorgement and the upper part anaemia, whereas both parts may have been perfectly normal during life. *No swelling (or projection) of the nerve head is observed in frozen sections of the dog's eye.*

On ophthalmoscopic examination of the fundus oculi of the living dog, the disc is seen to be oval (sometimes triangular) in shape. An

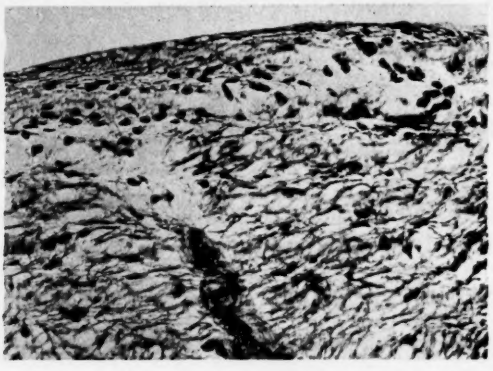

FIG. 13.

High power view portion of normal nerve head of dog to show the pseudo-oedema (vacuolation) that takes place if fixative is 4 per cent. of formalin in distilled water.

outstanding feature of the canine disc is the almost complete ring of large veins on it. To this venous ring the retinal veins converge, and as they pass across the edge of the disc they do not lose their light reflex. The arteries are all cilio-retinal, so that no true arteria centralis exists. (Fig. 14.)

The ciliary vessels pierce the sclera immediately around the optic nerve and pass inwards to the nerve head just in front of the subarachnoid space, where they give off the retinal branch. This enters the nerve at the level of a ledge of retino-choroidal pigment (usually present in the dog) to appear at the periphery of the disc. A central retinal vein (or veins), on the other hand, is often present but is exceedingly short, in fact it leaves the nerve head where the arteries enter it and then passes in front of the subarachnoid space. *In no case have we seen the main retinal vessels actually crossing the subarachnoid space of the dog* (Figs. 16-19).

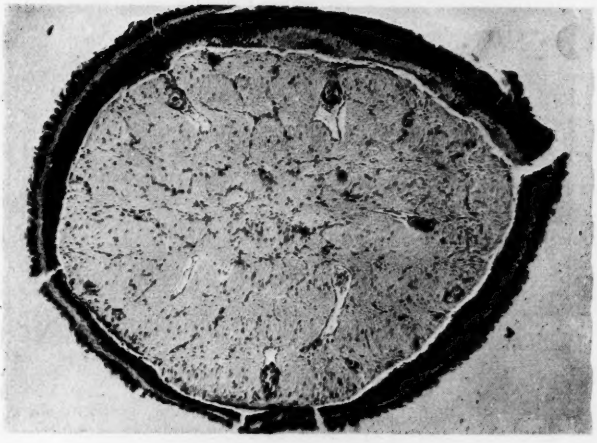

FIG. 14. × 70

Transverse section of canine optic nerve still within the globe to show cilio-retinal arteries (no central vessels).

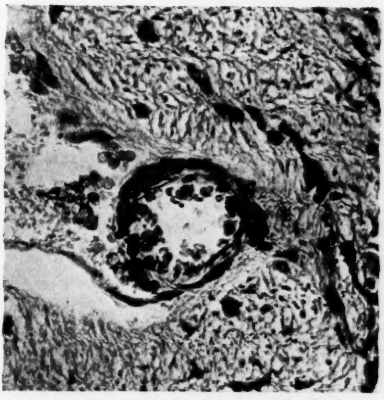

FIG. 15. × 500

High power view of a cilio-retinal artery in Fig. 14 to show *normal* pigment round the vessel which gives rise to a bluish halo when seen with the ophthalmoscope.

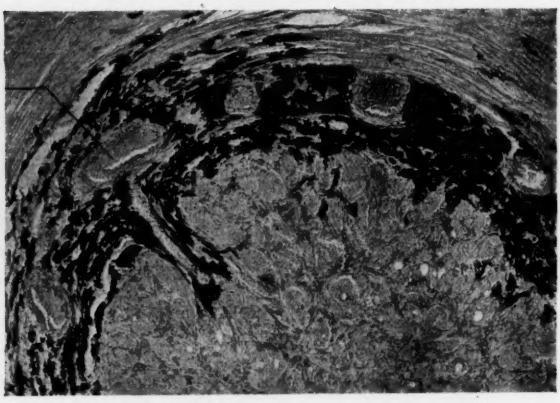

FIG. 16. × 75

Transverse section of canine optic nerve still in the globe, to show cilio-retinal vessels outside nerve with one C.R. entering it.

FIG. 17. × 60

Longitudinal section of canine optic nerve to show cilio-retinal vessels entering nerve at level of ledge of choroidal pigment. Note subarachnoid space further back.

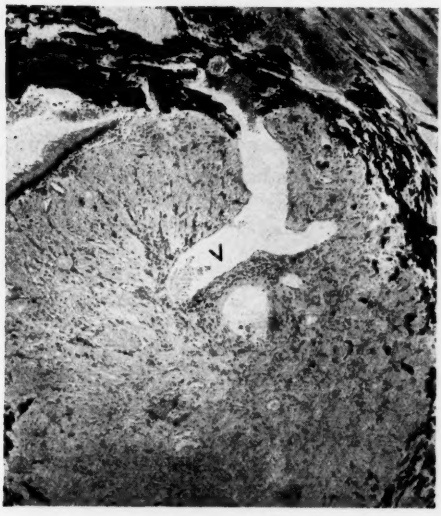

FIG. 18. × 75

Transverse section of canine optic nerve still in the globe to show main retinal vein V leaving the nerve.

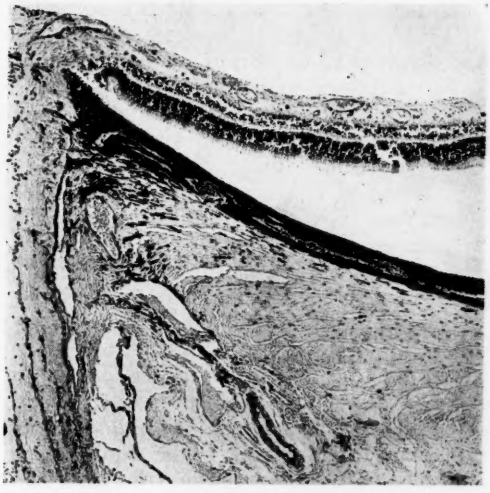

FIG. 19. × 75.

Longitudinal section of canine optic nerve to show cilio-retinal vessels passing in front of subarachnoid space.

The arteries are very much smaller than the veins and it is often difficult to follow their light streak as they pass across the edge of the disc. Hirschberg explains this as a contrast phenomenon due to the green colour of the tapetum. Under the best of circumstances the light streak on the arteries is narrow and not at all easy to follow. In many cases the edge of the disc is not very sharply defined, a fact also noted by Schieck (14 *c.*), and this circumstance makes it difficult to assess slight changes in it.

With regard to the possibility of producing papilloedema by injecting fluids under pressure into the cranial subarachnoid space, the results of the following workers may first be considered. Manz,[30] (1870), stated that injection of fluids under pressure into the cranial subarachnoid space in rabbits produces dilatation and tortuosity of the retinal veins and hyperaemia and swelling of the optic nerve head. This author's results are often quoted but they have been criticised by von Schultén,[19] and it may be noted here that these experiments were performed on animals most of which were not anaesthetised, whose pupils were not dilated, and whose eyeballs rolled about during the observations. It is impossible to assess fine changes in the retina under such conditions. von Schultén[19], (1885), found that raising the pressure in the subarachnoid space in rabbits resulted in narrowing of the retinal arteries and dilatation of the retinal veins, but never in swelling of the disc. Merz,[31] (1900), on the other hand, stated that in dogs, raising the intracranial pressure 15 to 20 mm. of mercury produces typical papilloedema. Kampherstein and Heine,[32] (1905), however, repeated Merz's experiments and found that they could not produce papilloedema, at least so long as the animal was alive. (Some of these experiments were performed under the supervision of Uhthoff.) Adamkiewicz,[22] (1895 and 1905), working with rabbits, found that injecting coloured fluid (Berlin blue saline) under pressure into the subarachnoid space produced no change in the fundus oculi until just before death, when the vessels disappeared and the disc went white. If the raised pressure was maintained after death, the coloured fluid entered the choroidal veins. Cushing and Bordley[20], (1909), experimenting with dogs, state that by injecting fluid at a pressure of 30 mm. of mercury into the cranial subarachnoid space, a swelling of six dioptres was observed in the disc in eleven minutes (observation 6). In another experiment (observation 5) they made a one-inch trephine hole in the midline, just anterior to the occipital protuberance, thus exposing the superior longitudinal sinus and a portion of dura over each cerebral hemisphere. A few minutes' pressure exerted with the finger against the exposed dura sufficed to produce a swelling in the disc of two dioptres. A stronger

pressure continued for some time increased the swelling to seven dioptres! Schieck,[14] (1910-1911), is of the opinion that dogs, cats and rabbits are unsuitable for these experiments as they have optic discs whose edges are normally ill-defined. This worker produced papilloedema by this method in apes and dogs, the latter being used only as controls. Contrasted with Schieck's results, Levinsohn,[23] (1912), failed to produce definite papilloedema in apes, dogs and cats by the above means. von Hippel,[33] (1923), reviewing the above experiments, is at a loss to find a satisfactory explanation of the contradictory results.

Of the above workers, only Cushing and Bordley reproduce a microscopic section showing distinct swelling of the optic disc, but the method of histological preparation of this section needs careful consideration. The authors state that with the object of fixing the oedematous retinal tissues under the conditions of the experiment so as to obviate in a measure the inevitable shrinkage of subsequent fixation, a solution of 4 per cent. formalin (? 4 per cent. formalin; ? 4 per cent. formaldehyde) was employed instead of salt solution for the injection fluid. The eye was removed post-mortem. The formalin used was obviously not strong enough to fix immediately the living tissues with which it came into contact, for the animal lived for one and a half hours with the fluid presumably in contact with the medulla. Even if the formalin entered the optic nerve head (and our injection experiments showed the diffusible dyes did not enter the disc) it would undoubtedly be only strong enough to act here as an irritant to the living tissues and may quite well have produced oedema in this way.

In many of our experiments, in which we kept the pressure of the injection fluid about 70 mm. of mercury, we could not convince ourselves that any visible change occurred in the fundus oculi, although post-mortem we always found the subarachnoid space around the optic nerve distended with dye. In some cases we thought that there was possibly some dilatation of the retinal veins. Slight changes in the arteries are very difficult to judge since they are normally narrow. In some cases the pressure of the injection fluid was gradually raised and it was observed that when the pressure reached 100 to 200 mm. of mercury (varying with different animals) the animal would stop breathing and the heart's action would become so weak that the retinal arteries practically disappeared and the fundus became pale and the disc dead white. The pressure would then be lowered (by releasing the clip on the tube leading from the large bottle) and the animal gradually commenced breathing. Sometimes artificial respiration had to be resorted to in order to re-establish respiration. The retinal arteries rapidly reappeared and the colour of the fundus and disc soon returned to normal. In most of our experiments we noted that,

not long after the commencement of the experiment, the disc appeared to become hazy. At first we thought that this was due to swelling of the disc, but we soon observed that this appearance resulted from small areas of clouding of the cornea which are liable to occur under experimental conditions and are no doubt due to exposure. Owing to refractive changes which they produce they may give rise to all kinds of distortion of the disc. It was noted that when the disc was looked at through another (unclouded) part of the cornea, the disc appeared normal. von Schultén's[19] words are appropriate here: "(as) I know the difficulties of estimating the value of what is seen with the ophthalmoscope and the part fancy may play therein . . . . " These observations evidently indicate that it is unsatisfactory to regard simple blurring of the edges of the disc as proof of the presence of papilloedema. The criterion we employed in the diagnosis of swelling of the disc was disappearance of a previously present light reflex on the veins as they crossed the edge of the disc. The light reflex on the arteries, as aforesaid, is normally very difficult to follow. The light reflex is the image of the source of light (used in the examination of the fundus) which reaches the eye of the observer through the pupil. It is obvious therefore that the slightest swelling of the disc would cause a bending in the vessels and thus throw the image of the source of light beyond the pupil so that the bent portion of the vessel would appear dark (*i.e.*, it would lose its light reflex). Clouding of the cornea, although it may produce a blurred appearance of the disc, does not lead to a vessel losing its light reflex in one place while retaining it in another. It is remarkable that, in the extensive work that has been carried out on the ***experimental*** investigation of the method of production of papilloedema, this essential sign (loss of light reflex) has apparently not been employed in the diagnosis of swelling of the nerve head. Using this criterion in the diagnosis of swelling of the disc, in none of our experiments, in which the pressure of the injection fluid was kept at 70mm. of mercury for two to three hours, did we observe papilloedema.

In one case only did we see the disappearance of the light reflex. Here, however, the pressure had been put up so high that the animal stopped breathing and its* heart's action almost ceased. The vessels in the disc disappeared and the disc became quite white. On reducing the pressure to zero and after artificial respiration had been resorted to for some time the animal recovered. At first the disc went pink with each inspiration and white again with each expiration, and then finally regained its colour. It is possible that here

* Mr. Wilfred Trotter tells us of an almost exact human equivalent of the above experiment. Those cases who, apparently dead, have recovered after cardiac massage, often die subsequently, and on post-mortem examination, an *oedema of the brain is found*. Up to the present we have not been able to ascertain whether papilloedema was diagnosed during life in these cases.

we unconsciously repeated Starling's classical experiment on the production of oedema of the legs. He tied the femoral vein, no oedema resulted. He then tied the femoral artery, and when after some time he released it, oedema was produced. The cutting off of the blood supply had injured the endothelium of the capillaries and when the pressure in them become normal again they allowed an excessive amount of fluid to pass through their walls and oedema resulted.

It is possible that in our experiment the blood supply from the capillaries was cut off for a sufficient time to injure their endothelium and when the pressure came back to normal an excessive amount of fluid passed through their walls as occurred in Starling's experiment.

It is interesting to note that, as long ago as 1887, Deutschmann,[35] in criticising von Schultèn's results, stated that the failure of the latter to produce papilloedema experimentally was due to the fact that he had not cut off the *arterial* supply to the retina.

We have seen that it is possible to get only a non-diffusible dye into the eye at pressures incompatible with life. Moreover, the dye is found in definite vessels and not in the tissue spaces where one finds the oedema in papilloedema. Also we could not produce a coloured papilloedema either with diffusible or non-diffusible dyes. It would thus appear that, at any rate, in the dog, there is no communication between the peri-optic subarachnoid space and the optic nerve.

With regard to man the only evidence of a communication is the solitary experiment* described by Schieck. What seems certain to us, however, is that whether the papilloedema is produced directly or indirectly by the cerebrospinal fluid there is no reason why the whole of that part of the retina drained by the central vein should not become oedematous. If we take it that the vein is obstructed in the intervaginal space then the tendency to oedema must be the same in the whole area which it drains; the slight venous anastomosis at the entrance of the nerve would in fact, as suggested above, tend to carry the oedema fluid away and therefore make it less likely for the area to become oedematous.

We believe, then, that in papilloedema the entire area drained by this vein does contain an excess of fluid (oedema), but that the amount of fluid in each region of the retina will depend upon the density† of the structure of the particular region under consideration.

*We have not been able for the moment to repeat this experiment, for the opportunity of getting a freshly excised *normal* human eye with a length of optic nerve attached does not frequently arise.

†If a rubber tube which consists of alternating thin and thick portions be taken closed at one end, laid on its side and then gradually filled with water, it is obvious that the thinnest portions would bulge most, although all parts are under the same pressure. In exactly the same way all the tissue spaces drained by the central retinal vein must be under the same pressure; with increase of this pressure the amount of swelling must therefore depend entirely on the structure of the part.

Thus, the nerve fibres at the disc have a relatively loose arrangement and much fluid could collect here and produce obvious swelling, whereas the lamina cribrosa, with its dense meshwork of fibrous trabeculae, would, with the same capillary pressure, contain only very little excess of fluid. In papilloedema it has been noted that the swelling extends in the nerve fibre layer for a short distance only beyond the disc. This, we would suggest, is due to the arrangement of the fibres of Müller which hold together the elements of the nerve fibre layer. The Müllerian fibres in the immediate vicinity of the optic nerve head are stretched or torn (Paton and Holmes[5]), whereas those farther from the disc remain intact and limit the amount of fluid exuded. The remainder of the retina also undoubtedly contains excess of fluid, although usually not sufficient to be visible with the ophthalmoscope and, as aforesaid, may to a large extent disappear on microscopic section. The swelling of the disc (as opposed to the rest of the fundus) in cases of papilloedema appears to be parallel with that in the facial subcutaneous tissues in cases of renal disease, in which the lower eyelids swell, not because their capillaries differ in structure from those elsewhere, but because laxness of the tissues of the eyelid allows a greater accumulation of fluid which exudes from the capillaries. It is the structure of a part which determines the amount of fluid which collects in it (for a given capillary pressure) and therefore the amount of swelling which will take place in that part.

Finally we have to seek for a reason why the swelling of the disc in cases of papilloedema should appear at one part of the disc before another. Paton and Holmes[5] describe a fibrous band which would tend to keep the outer side of the disc strapped down in cases of papilloedema. The present writers have no knowledge of such a band, nor could any other reference be found to it. Schieck,[14] who believed that the subarachnoid space around the optic nerve communicated directly with the retinal lymphatics, and that papilloedema was due to cerebrospinal fluid being forced into the retinal lymphatics, was of the opinion that the swelling started where the large vessels cross the edge of the disc. Our experiments, however, demonstrated that there is no direct communication between the subarachnoid space and the retinal lymph spaces, at any rate in the dog, and thus Schieck's explanation of the special site of commencement of swelling of the disc cannot be upheld. The structure of the disc appears to us to afford a clue to this question. A study of the relative thickness of various parts of the nerve fibre layer at the edge of the disc in the human, shows that it is thinnest directly outwards, *i.e.*, in the region of the papillo-macular bundle. Next in thickness are the upper and outer quadrant and the lower and outer quadrant, then the innermost part of the edge of the disc, and finally the thickest parts are the

upper and inner quadrant and the lower and inner quadrant. The swelling will obviously first be visible in the thicker parts of the edge of the disc, and from the structure of the disc we would deduce that the order of appearance of swelling will be first, the upper and inner quadrant and the lower and inner quadrant, next the inner edge of the disc, then the upper and outer quadrant and the lower and outer quadrant, while the last part of the disc to show visible swelling will be the direct outer part.

In conclusion, our experiments are in accord with the general result of Weed's[36] work. This observer maintains that the subarachnoid space is a closed space.

Summary and Conclusions

1. Non-diffusible dyes injected into the cranial subarachnoid space at pressures compatible with life, do not enter the optic nerve.

2. The claims of previous investigators to have produced papilloedema by the injection of fluids into the cranial subarachnoid space at pressures compatible with life, are not upheld by the present study.

3. Anatomical observations are made on the normal structure of the optic nerve head in the dog and in man.

4. Structural reasons are advanced for the special site of commencement of papilloedema in man, and for the extent of distribution of the oedematous fluid associated with papilloedema.

Acknowledgments

The present investigation was mainly carried out in the Anatomy Department, University College, London, and completed in the Anatomy Department, King's College, London, and the Pathological Department of the Royal Westminster Ophthalmic Hospital. We wish to express our gratitude to Sir John Parsons, Mr. Percy Flemming, Professor G. Elliot Smith, and Professor D. M. Blair, for their interest and advice in the work. This study was made possible by a grant from the *British Journal of Ophthalmology*.

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THE MICRO-ORGANISM OF INDIAN SUPERFICIAL PUNCTATE KERATITIS

BY

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THIS note is somewhat overdue on the question of the pathogenic agent responsible for the widespread and persistent epidemics of punctate keratitis seen in India. Presumably the same specific organism may account also for isolated cases of similar disease seen not only in India, but also in other parts of the world.

The question was raised afresh as part of the admirably comprehensive and far-reaching clinical, pathological and bacteriological investigation of the disease, by Lt-Col. R. E. Wright and co-workers in Madras, published in the *Brit. Jl. of Ophthal.*, Vol. XIV (1930), pp. 257-282. It will be remembered that experimental evidence was there given pointing to a filter-passing virus as the infective agent of the disease, linking it with herpes simplex of the cornea in its varied forms.

Wright and I are agreed that the disease investigated in Madras is essentially the same as that of my old Bombay reports, published in *The Ophthalmic Review*, Vol. XX (1901), pp. 339-345, and in *The Ophthalmoscope*, Vol. II (1904), pp. 8-9. There is no doubt also that Kirkpatrick's account in the *Brit. Jl. of Ophthal.*, Vol. IV (1920), pp. 16-20, dealt with some phases of the same epidemic disease.

How is it that in all the 30 years since my first report no widely known ophthalmologist has endorsed my finding of a causative encapsuled bacillus, except the late Prof. Axenfeld, to whom I sent a number of slides, stained and unstained? Apparently no one in India has done any work at all on the origin and spread of the disease since my efforts, except those above mentioned, and I feel sure that the failure of the Madras workers to find the organism that I had described is mainly due to my failure to emphasize the extremely limited distribution of the organism in, or on, the corneal epithelium. I hope that it is still not too late to induce someone in India, with the requisite facilities and opportunities, to utilize and to extend the following observations.

The bacillus appears to be confined entirely to the more active points of the disease, as defined by fluorescein staining, and its very feeble staining qualities add greatly to the difficulty in finding it. The search was found to be altogether too tedious unless confined solely to cases of the disease presenting the more closely packed, or the larger staining spots.

The work was certainly facilitated by the use of a stronger solution of fluorescein than the usual one. A saturated solution was

made in a rather stronger solution of sodium bicarbonate than was laid down (I forget the precise strength). Two or three instillations were made into the patient's eye, and their effect seemed to be enhanced by an intervening cocaine instillation, restricting lacrimal secretion.

The selective colouring of active points thus secured was distinctly more effectual, and more useful, than with weaker fluorescein solutions. The aid thus given was particularly noticeable in some of the " disciform " phases of the disease, presenting a rounded central area of diffuse haziness, with defined raised margin.

The closely packed bacilli appeared to lie exclusively at the surface of the epithelium, in a single layer or little more. Their total number was evidently quite small, compared with disease of uniform distribution. Accordingly, in taking epithelial scrapings for examination light touches were made at the stained spots with the point of an old broad needle, well blunted by repeated passage through the flame, and the scanty material was well broken up on a moistened slide.

One could only hope to find one or more groups of organisms lying on, or in epithelial cells, among a large number of bacteria-free cells. Doing the whole of the work myself of collecting, fixing and staining the material, I was able in early days to estimate only " two or three " complete failures to find the bacilli in the examination of at least two or three dozen eyes. If there had been unlimited time at my disposal, probably there would have been no failures.

The most certain stain for determining the presence of the bacilli was by Gram's method, with incomplete differentiation, leaving a good deal of the tissue much overstained, and without the use of a counter-stain. But the characteristic features of the organisms were not shown thus. The capsules were not sharply outlined at all; apparently they stained more or less with the central portions. Unfortunately, I failed to seize the opportunity for closer study of the bacillus by the use of varied counter-staining after the Gram stain. The most characteristic results were obtained by the use for 10 or 15 minutes, or more, of a pale diluted borax methylene blue solution, followed by a similarly diluted eosin solution.

The colourless capsules were then sharply defined from their pink surroundings, and easily measurable. The much less defined blue centres varied in size with the depth of the blue staining. A strong impression was given that the sharp-edged capsule represented an integral part of the organism, differing essentially, for instance, from the very variable capsule of the Morax-Axenfeld bacillus.

Dr. Surveyor, of Bombay, gave the average size of the bacillus, defined by the outer edge of the capsule, as $1{\cdot}6\mu \times 3{\cdot}2\mu$. Axenfeld gave practically the same measurement of the bacillus, viz. $1{\cdot}5\mu \times 3\mu$. It is unthinkable that this universally recognized authority and acute observer should have accepted the aetiological significance of the organism as " very probable " without good reason. In his illustrated description in " The Bacteriology of the Eye," MacNab's translation, 1908, pp. 325-6, he remarked that "these quite peculiar bacteria . . . were only stained with great difficulty . . . in the ordinary stains were merely hinted at, and were only demonstrable on account of their well-defined capsules."

To sum up, I personally have no doubt whatever that the bacillus is the living infective agent of the disease. I believe that it is always present in the active phases of the keratitis, and, so far as I know, it seems to be a unique organism, with unmistakable features. Its distribution—its presence in relatively large numbers at the stained spots, and there only—cannot be readily explained away.

What is the present position? How can the above findings and statement be reconciled with the Madras experimental production of the disease in human eyes in five out of eleven attempts, by rubbing in *filtrates* of epithelial emulsion obtained from diseased corneae, usually after scratching the epithelium of the eyes upon which the implanting was to be done? The proportion of successful implantations in these attempts with filtrates was the more noteworthy, in that it was not lower than was attained in a smaller series of similar attempts (three successful in a total of seven attempts on unaffected human eyes) made with *unfiltered* epithelial suspension from diseased eyes. The presumption of an active living filterable virus seems very strong from these results, and yet it must be wrong if the natural deduction from my work is to be accepted.

What is to be the outcome of this conflicting evidence? The solution of the problem calls for further investigation in both directions. In the first place renewed search for the encapsuled bacillus should be a simple matter if the disease is still epidemic in India, or when it returns. If material is available I am quite sure that any really careful and determined bacteriologist can find the bacillus as regularly as I did, using the precautions and technique given above. And something more might be learnt about the organism.

Some facts bearing upon the problem may be briefly reviewed.

There has been remarkably little evidence of transmission of the disease from patient to patient during epidemics. Do not the strongly-defined capsules suggest an exceptional resistance of the organism to drying, in its existence away from the human eye?

A natural existence apart from the eye is perhaps suggested also by its failure to penetrate deeply into the corneal epithelium. Can the widely-spread disease be largely air-borne? An obvious argument against this is the comparative rarity of binocular affection. But this difficulty is to some extent overborne by the recognition that some slight traumatism, or some slight conjunctival irritation or conjunctivitis may be largely instrumental in implanting the infection. Wright says "Trauma is noted in the history of a large number of cases and it is quite likely that it favours an attack." In my earlier report the fact was noted that in the only case of well-marked simultaneous double punctate keratitis that had been seen, subacute trachoma was present also.

It is necessary to recall here my one experience in experimental production of the disease, briefly mentioned in *The Ophthalmic Review*.

At one time cultures of bacillus pyocyaneus were apparently grown in Bombay from some eyes affected with the keratitis. So far as we could do so, we excluded the possibility that these growths—very unusual from corneal surfaces—may have represented "outside" contaminations. One of the cultures was implanted on the everted upper lid of a hospital attendant. This produced a couple of small pustules followed by a curious greyish persistent palpebral conjunctival patch.

Six weeks after the implantation the cornea of this eye was found to have a mild attack of punctate keratitis. It was only a very poor development, recognizable at first by two typical staining points only. But it was undoubtedly the real thing, no less developed than in many eyes seen regularly in the out-patient department. It may have existed for some time unnoticed.

Whatever may have been the exact relationship between the pyocyaneus implantation and the subsequent slight keratitis, there is little doubt that the connection was in some way, and in some degree, an aetiological one, since this was the only eye of anyone connected with the hospital, that was ever attacked by punctate keratitis in my experience.

Apart from the remote possibility of a symbiotic relationship between the b. pyocyaneus and the encapsuled bacillus of punctate keratitis, the incidents related may be regarded perhaps as merely another example of trauma or conjunctivitis "favouring an attack" of the keratitis during a widely prevalent epidemic. That is, the attack probably presented merely a slight variation from many of those seen regularly among the out-patients at that time.

But this limited success, such as it was, of our single experiment prompts the inevitable inference. In how many of the more strikingly successful Madras experiments may not the same explanation have sufficed fully and truly? Can it possibly have

been the true explanation in all? The rapid and early developments in the successful Madras cases may be held to correspond merely with the direct corneal traumatism inflicted in these experiments—scratching and massage.

Controls appear to be needed consisting of exactly similar traumatism at an epidemic period, without the use of any epithelial emulsion from diseased eyes, but possibly with the application of emulsions, filtered and unfiltered, taken from healthy eyes.

OCULAR PLUMBISM IN CHILDREN

BY

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(SUMMARY.—The cases dealt with in this paper have no pyrexia. The mild ones are brought by their mothers because they have developed a recent internal squint. The squint is due to paralysis of one or of both external recti. Papilloedema up to six dioptres is found, due to increase of intra-cranial tension. The severe cases are more evidently cases of lead encephalopathy. Explanation is given of why soluble lead is available, and why and how it is ingested. Lead is found in the urine. No albumin is found in the urine.)

ALTHOUGH I have written on this subject in Australia, I have only published one paper in England, and my knowledge has increased and become more accurate since then.

The peculiar nature of the cases described has not as a rule been thoroughly recognized. They appear to have no parallel among cases of industrial lead poisoning, and nearly all writers on lead poisoning where the eyes are implicated, have had experience of industrial lead poisoning only, and of that in adults. My reason for frequent references to the cases I have seen in Queensland has been the desire of myself and of my colleagues, especially of those at the Hospital for Sick Children, to get a danger removed from our young children between the ages of two years and eight years; and also to make isolated cases in other places and other countries less likely to be undiagnosed as due to lead—to bring lead at least into the differential diagnosis of such cases in young children.

It has been difficult for those practising in other parts of Australia to recognize the special conditions under which our children live, and the special facilities they have for ingesting lead. It has proved quite impossible for many of those who have investigated industrial lead poisoning to grasp the fact that whereas industrial lead poisoning occurs owing to the inhalation of lead dust, our children are poisoned owing to the ingestion of

powdered soluble lead carbonate. The explanation given of how this soluble lead carbonate is ingested, and the reason why such poisoning among children is less common, or absent, elsewhere than in Queensland, industrial lead investigators are unable to appreciate.

The British Medical Association, Queensland Branch, succeeded after many years in getting the Government to make it penal to apply lead paint to outside surfaces within the reach of young children. This law has been in force since 1923, though not always obeyed by master painters. It, and still more the persistent teaching of parents by those of us who grasp the importance, has resulted in many fewer cases of hand and foot drop, and of ocular plumbism.

The ocular plumbic cases, if secured early and treated promptly, will recover without any defect in sight. I am personally answerable for claiming them to be due to lead, for claiming them to be a result of increased intra-cranial tension, and for the explanation that the lead circulating in the vessels of the choroid plexus causes the increased secretion of cerebro-spinal fluid.

The frequency of wrist drop and foot drop cases, which were readily diagnosed as due to lead, caused me to diagnose the eye cases. I am sensible that it would have taken much longer to arrive at this conclusion in the eye cases had it not been for the wrist and foot drop cases. The two conditions very rarely occur together. Latterly, I have been able to explain this, and Woollard's work with dyes has supported my explanation in an interesting manner, as I will explain later.

We were able to diagnose the limb cases and the eye cases for years before I was able to discover how and where they ingested their lead.

In Queensland, houses are built on piles. The verandah floors and other floors are therefore several feet above the ground. All our dwelling houses, which are chiefly wooden, have verandahs more or less broad. They require verandah railings. These railings are of wood, and in the neighbourhood of towns they are painted. Except where other paints are insisted upon, they have been painted with white lead paint. In this semi-tropical climate, the sun soon converts this paint into a powdery substance. The powder rubs off easily on the hands. This powder our Government analyst in 1904 proved for me to be a very soluble carbonate of lead. Garden fences are painted with the same material. Such paint inside our houses or unexposed to the sun and outside atmosphere, does not powder at all appreciably. The children between the ages of two years and eight years spend a large part of their time on the verandahs, girls more than boys. They are apt to finger and to cling to the verandah rails. I soon

found that the children in a family who become affected with lead bite their nails, and the few who do not, suck their fingers, or occasionally suck the paint. It was found that more girls were affected than boys; that more cases occurred during the summer months than in the cooler months, because the moist hands of children during the hot months carry more of the lead powder to their mouths than the dry hands of winter.

The cases of wrist drop and foot drop were first described by the staff of the Hospital for Sick Children in 1892. I was then an Honorary Physician. Since then, Dr. A. Jefferis Turner especially, and other members of the staff, have published papers. The eye cases I have recognized since 1897, when I became Honorary Ophthalmologist to the Hospital. At first they were named by me ocular neuritis due to lead, and thought to be due, like other lead cases, to peripheral neuritis. Latterly, I have regretted this name, because ocular plumbism would be more accurate.

Continued observations and treatment revealed the existence of increased intra-cranial tension in the ocular plumbic cases, and that the papilloedema and paralysis or paresis found in the external recti muscles was due to this increased intra-cranial tension. It was found that if the cases were admitted, *i.e.*, removed from the opportunity of ingesting more lead before the increased intra-cranial tension had lasted for long, a prompt lumbar puncture, followed, if necessary, by a second lumbar puncture in three days, very quickly resulted in reduction of the papilloedema and of the squint. I say " resulted " because we had been able to contrast what happened after I had insisted upon lumbar puncture becoming a routine practice with what happened when I did a lumbar puncture only in cases with marked head symptoms in addition to the papilloedema and squint.

I am, of course, conscious that the ocular plumbic cases are cases of lead encephalopathy, but to give them so high sounding a name might defeat my object, which is to get practitioners elsewhere than in large cities to deal with them promptly when a doubt exists regarding the aetiolgy. The removal of the child from further possible contact with powdery lead paint, and the performance of a prompt lumbar puncture, is the essential treatment, *i.e.*, remove the cause or further cause, and reduce the pressure, which will destroy the sight if it lasts long enough. (The amount of fluid removed on the first puncture varies from 2 drachms to 18 drachms.)

The early cases hardly appear to be ill, and would be unrecognized by their parents except for the sudden and fortunate development of a squint. The severe cases are, if undiagnosed, again fortunate in being so sick that they are confined to bed and so removed from further ingestion of lead. This limits the damage to

vision, but as a rule results in complete or partial post-papilloedemic atrophy in one or both eyes unless a timely lumbar puncture is done.

The Government analyst, after taking every precaution to exclude contamination of specimens and reagents, found lead in sufficient quantities in the urine of the children to prove the absorption of lead. What the faeces contained had not necessarily been absorbed from the intestinal canal, and was not therefore of importance.

Occasional cases similar in all respects occurred when children had been allowed access to fresh paints, either on recently painted houses or in pots of paint used by their fathers in the house. The following case is one in point:—

October, 1913.—H. H., male, aged 5 years 8 months; sent to me by Dr. Coen, of Lismore, New South Wales. The illness began a month ago with vomiting without relation to food and lasting ever since except for three days intermission; frontal and temporal headache; internal squint noticed for two weeks, and double vision.

Examination: Child very pale. Paralysis of each external rectus and a very marked internal squint. Eating nothing to speak of until the last two days. Temperature normal. Intense papilloedema (choked disc) in each eye. Discs raised six dioptres; veins very distended; retina surrounding each disc swollen; no head retraction; said to see well—can see my fingers at six yards. Nails are all bitten.

The house has no verandah railings, but the garden is surrounded by railings painted with white lead five or six years ago, and now very powdery. His father does some painting and mixes white lead paints in the house. He has been mixing some lately, specially for a month before the boy became ill. Since the boy has been sick, he has been too ill to go near either his father's paints or the garden railings. He had been decidedly better for the last two days, his mother told me. Family history excellent. I gained the impression that he was beginning to show benefit from being too ill to play with his father's paints, and therefore to continue the ingestion of lead.

Ocular plumbism was the provisional diagnosis. An immediate lumbar puncture was done. The cerebro-spinal fluid was under pressure, but not to the extreme degree often found in lead cases and only two drachms of fluid were withdrawn. (No doubt the extreme pressure was subsiding.) He slept well that night, and his headache disappeared and did not return. The right external rectus showed some return in power next day. In four days the external recti were acting fairly well and there was little squint. In five days there was only two dioptres of swelling in each disc. In 14 days only a trace of alternate squint persisted and the papilloedema had subsided. He had been given, of course, dilute sulphuric acid and magnesium sulphate, followed after the first week with potassium iodide. He was allowed to return to his home in the north of New South Wales.

In August, 1929, *i.e.*, 16 years later, a broad shouldered, particularly healthy-looking young man—a typical footballer—called to tell me how grateful he and his parents had been to me. His vision was six-fifths with each eye. Each fundus was natural, and refraction emmetropic. One had to imagine that the slightly irregular edges of the discs may have been a legacy of the papill-oedema. There was no sign of any ophthalmoplegia.

The ocular plumbic cases being due, as I think, to the ingestion of a considerable quantity of lead during a short time—a few weeks—would not be expected to lead to the serious sequelae which chronic lead poisoning in childhood is supposed by some of my physician colleagues to be answerable for in young adults. Dr. Nye

has, indeed, made out a very good case for the unusual number of nephritic young people in Queensland being a result of ingestion of lead in childhood. This case and others help to confirm the aetiology of the more usual eye cases. The hand and foot drop cases are almost always a result of prolonged ingestion of small quantities of lead. The ocular cases, I believe, ingest considerable quantities over a short time. This explains what has always been a difficulty—why some children get hand and foot drop, and others only ocular plumbism. Why more massive doses over a short time cause eye symptoms may, I think, be explained by an observation of Woollard, who found that if small doses of dyes are given, no head symptoms result, because apparently the cerebral circulation is fenced against them. If, on the other hand, large doses are given, head symptoms and convulsions occur. I believe that the lead circulating in the choroid plexus causes an increased secretion of intra-cranial fluid and consequent increased intra-cranial pressure, with resulting papilloedema and paralysis or paresis of the external ocular muscles, nearly always of the external recti. I have known all the ocular muscles implicated in a severe case brought too late to prevent blindness. The squint is always recovered from.

As said before, my chief object in again writing on this subject is the hope that it may lead to isolated cases being at least provisionally recognized and treated, and blindness amongst children thereby reduced. I think also that my explanation of how and why papilloedema is caused is original and correct.

We have found that a good substitute for lead paint is zinc white. When used without admixture with lead, it lasts on exposed surfaces longer than lead. It powders less, and its powder is not poisonous. I have had only one voluntary explanation as to why master painters resist wholeheartedly the use of any paint whose basis is not lead. The senior painter in Brisbane said to me when I first began to urge zinc as a substitute: "You know, Doctor, we painters do not like this zinc craze." "Why, Mr. . . . ." I asked. "It lasts too long," came the answer. Those interested in white lead manufacturing resist for other reasons also.

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END RESULTS IN MONOCULAR ESOTROPIA*

BY

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OPINIONS are divided as to what may be expected as end results in monocular squint. Many believe that only parallelism can be obtained in the great majority of patients, with an exceptionally good termination now and then, including equal visual acuity in the two eyes and, to all intents and purposes, single binocular vision. In fact, not many years ago a member of this Society, an unusually good operator, expressed his belief in a paper that comparatively few children recover from squint with single binocular vision. There is, however, a minority of operators, in which the speaker is included, who believe the ideal goal can be obtained in the vast majority of young squinters, *if systematic measures are instituted, if the squint is treated at the proper age, and if the co-operation of the parents can be obtained.*

It might be well to state just what the ideal goal embraces in order to say "the results are ideal." In my judgment, ideal results include, first, good visual acuity in the squinting eye; second, single binocular vision which will stand the proper tests;

* Read by invitation, at the Annual Meeting of the Pacific Coast Oto-Ophthalmological Society, May 29, 1931, Los Angeles, California, U.S.A.

third, good convergence for sustained close work; and fourth, comfort for the patient, which is necessarily predicated on a good muscle balance after treatment is completed. In order to obtain such results, each of the four conditions just mentioned plays an important rôle, but for obvious reasons they may not all be obtained in a given case. So long, however, as the break in the results does not occur because of our own misjudgment or neglect, there should be no laxity on our part to strive to obtain these ideal results in every case. To assume, however, that one can lay down certain definite procedures which, if followed, will bring about the desired goal, is begging the question. The object of this paper is to endeavour to establish these facts.

Factors which interfere with Ideal Results

Our purpose will be served best by reviewing, first, those facts which, if present, will definitely interfere with perfect results; and second, by analyzing the factors which have yielded this ideal goal in many cases, and will continue to yield the same end results in many others to come.

Aside from the neglect on the part of parents, whether from ignorance or from indifference, the two technical conditions which stand as a barrier to a perfect outcome are amblyopia ex anopsia and an undeveloped or absent fusion faculty. The former, in all but a few instances, is remediable under proper conditions. As to the latter a fusion faculty, if totally absent, cannot be developed, but if present, even in moderate degree, can be awakened by proper methods. As to how these barriers can be removed will be discussed in the second part of the paper. For the time being, I wish to stress the important fact that in a majority of instances in which only cosmetic results are observed, failure to obtain ideal results can usually be attributed to the presence of one or both of these barriers.

Factors which favour Ideal Results

In constructing our framework upon which a perfect issue may rest, there is one feature which is thoroughly understood and accepted, and this will be passed over rapidly. I refer to refraction.

1. *Refraction.*—The first essential is early and careful refraction. This is accepted by all as fundamental in the management of squint. It offers a large field for discussion, but suffice it to say that one's duty to squinters is not completed with the careful fitting of glasses. It is apropos to add that in the vast majority of cases, the prevailing error is high hyperopia with anisometropia. The latter element, in my experience, is the precipitating factor

which paves the way for the failure of a weak fusion faculty to hold the visual axes in alignment, and for the subsequent development of squint.

2. *Prevention and Correction of Amblyopia ex Anopsia.*—In the course of treatment, the second factor which demands our attention—a factor which, in many instances in the very young child, is first in importance and in order of time—is the prevention or correction of amblyopia ex anopsia. In former communications, I have endeavoured to show that the amblyopia of squint is acquired in all but a few instances, and amenable to treatment under certain conditions.

Amblyopia ex anopsia is observed in three types of patients: First, in manifest monolateral esotropia of childhood; second, in adults who in early childhood had a manifest squint which disappeared spontaneously or by systematic treatment which may have included operation; and third, in adults in whom there is no history of squint in childhood. In these three groups, the characteristics of amblyopia ex anopsia are the same, save in the degree of amblyopia, and the dominating refractive error is that of anisometropia with prevailing high hyperopia almost universally present. The three characteristics of amblyopia ex anopsia, as pointed out in a paper before the last International Congress at Amsterdam, are: (1) A central, usually relative, scotoma about three degrees in diameter; (2) an enlargement of the blind spot of Mariotte; and (3) a moderate contraction of the form and colour fields. This evidence of lowered sensitivity can be demonstrated in almost all cases, and equally well can be shown to be the result of disuse and amenable to treatment. In all cases in which lowered central vision can be elicited by subjective tests, a central scotoma can be outlined. Those of you who have practised orthoptic training in young children up to the age of five years must be impressed with the ease by which the squint can be transferred from one eye to the other with lowering of central vision in the secondarily squinting eye. The younger the child, the more readily can the squint be transferred, whereas between the ages of four and seven, the transfer is less easily effected. I do not recall a single instance of transfer after the seventh year. What is equally interesting is the greater difficulty encountered in the entire correction of amblyopia in children over seven years of age.

According to Ida Mann*, the macula of the new-born does not take on tne histological appearance of the adult until 16 weeks after birth. Furthermore, it is also a well-known fact that the functional development of the nervous system of the child is not complete until the child is five years of age. These facts are sufficient to

* The Development of the Human Eye, 1928.

explain the instability of macular vision, and the ease with which central vision can be disturbed in young children.

An observation which is equally interesting, and as readily demonstrated, is the ease with which lowered central vision can be corrected in children up to four or five years of age, and the increasing difficulty in correcting the same with each succeeding year of life after this period. The fact that the treatment of squint is so often instituted after the seventh year is probably responsible for the unfortunate prevailing opinion of many that nothing can be done to correct the amblyopia, and the equally inaccurate observation that the amblyopia of squint is congenital and, therefore, a cause of squint. Nothing is further from the truth.

It will take us too far afield to enter into a discussion of the details necessary for the correcting of this amblyopia. Suffice it to say it can be done in the young child, and to a less degree after the fifth year. If corrected, it furnishes the second important step which will admit of ideal results in monocular esotropia. Although an important barrier to cure in monocular esotropia, a moderate degree of amblyopia, with vision of 6/9 or better, is compatible with good fusion and otherwise good results. The fusion areas in the retinae extend beyond the fovea and probably beyond the macula. It is this fact which aids in cases of spontaneous cure of squint, and it also is operative in amblyopia ex anopsia of adults who do not have a history of squint, even in childhood. I have observed the same fact in otherwise perfect cures by training and operation. If fusion can be awakened to activity, a moderate degree of amblyopia will not prevent a cure. Its prevention or perfect correction, however, materially hastens and assures perfect results.

3. *Training of the Fusion Faculty.*—The third measure essential to ideal results deals with the fusion faculty. After the third year, the state of the fusion faculty can be determined in many instances. In some children this cannot be estimated before the fourth year, and in a few instances not before the fifth year. Its determination, however, is vital in the management of squint. Although it is a *sine quâ non* in perfect restoration of single binocular vision, and in some instances associated with the first and second measures accomplishes the results without the necessity for an operation, its treatment, in some instances, may be deferred a year or two without seriously interfering with the ultimate goal. I refer in particular to those cases in which the squint is of such degree that an operation is necessary. Although any of the four measures mentioned accomplishes maximum results in young children up to five years, a child of seven is more pliable for the training of fusion. Good fusion is necessary for the prevention of squint, and in the final analysis, it is the keystone,

so to speak, of the arch which bridges the chasm between cosmetic results and the goal which is sought—single binocular vision. In my experience, if a fusion tendency is present but undeveloped, it can be awakened even in adult life. Because of this fact and the somewhat complex act required in its training, it can often be left to the age of six or seven years, if the younger child responds indifferently. In fact, in the second group of acquired amblyopias previously referred to (squint in early life with subsequent spontaneous cure), the results are brought about, in my judgment, by the self-awakening of the fusion faculty. Furthermore, the occasional brilliant results obtained after proper refraction and accurately performed operations, without special fusion training, are also the result of the self-awakening of a fusion faculty which previously was present, but in latent form, because it could not operate in the presence of wide deviation of the visual axes.

Without entering into a discussion of the nature of a fusion faculty, a fusion sense, fusion power, or any other term which is implied in single binocular vision, facts which are patent to all are the dominance of this faculty in all normal individuals, its total absence in true alternating esotropia, and its readiness to spring into activity in all cases of monocular esotropia when normal relations are restored by improving vision, by the prevention or removal of amblyopia ex anopsia, and by restoration of approximate parallelism of the visual axes. Something inherent in the individual tends to its normal return, but artificial stimulation hastens and restores it to normal function, and in many instances, becomes necessary to its full awakening.

4. *Operative Interference.*—In a large number of cases, the fourth and final step in the treatment of squint becomes necessary. The reasons for the necessity of this fourth step are to be found in high degrees of squint and in the molecular and organic changes in the muscles themselves, and in the capsule of Tenon. In high degrees of squint, the separation of the visual axes places the images beyond the reach even of normal fusion, and much beyond that which a feebly developed fusion faculty can bridge over. Anatomical changes which develop in the muscles, in their fascial sheaths and in the capsule of Tenon in well-established squint furnish a second barrier, which in many instances must be removed by operation. If the squint is of such degree, much is gained by an early operation. In fact, the longer one studies the various phases of squint, the stronger becomes the conviction that each of the four steps enumerated yields the best results before the end of the fifth year. In other words, if parallelism of the visual axes can be established in the early years—the formative period, vision is less endangered and the fusion faculty requires less

artificial stimulation. The fight against amblyopia ex anopsia must be kept up until parallelism is restored automatically, by consistent training, or by operation. If indicated at all, therefore, operation should be practised early.

The methods of operating are many. Each surgeon may have his own preference, but the patient's interests are best served by the choice of such procedures as are especially indicated in the individual case. We cannot stop to discuss the special advantages of the operations at our disposal. Only principles are of importance for our purpose. Basic principles which should be mentioned are the following:—

1. The converging power of the internal recti should be preserved. As a natural corollary, if it is necessary to operate on one or both internal recti, nothing but muscle recession should be practised. Tenotomies are contra-indicated.

2. If the squint can be corrected by shortening one or both external recti, the results will be most satisfactory and normal convergence will not be placed in jeopardy.

3. Supplementary operations, if necessary, should be reserved for the internal recti. There are occasional exceptions to this rule when the internus is definitely in a state of contracture.

4. Only such operations should be selected as will admit of the most accurate correction of the squint.

5. Although the cosmetic appearance of the bulbar conjunctiva is secondary in importance to the accuracy of the restoration of parallelism, it is worthy of serious consideration in the choice of operations.

No statistics are offered to assure you that the methods set forth will yield the ideal results which were stated in my premise. Statistics are rarely analyzed. I have been following this plan of procedure for many years, and can assure you that the end results obtained by this plan warrant the statement that single binocular vision, with good visual acuity and comfort to the patient, can be obtained in most cases of monocular esotropia during the first seven years of life. They can be obtained in older children in a smaller percentage of cases. The same systematic plan should be followed at all ages, and if followed, a nearer approach to ideal results will be obtained. The unbelief and scepticism as to such claims in monocular esotropia, which are prevalent but fortunately on the wane, are the result, first, of antiquated teaching, and second, of the fact that the average case of squint is treated too late to obtain anything but cosmetic results. If the family physician can be induced to refer his little squinting patient to the eye specialist as soon as the squint appears, and the measures advocated in this communication are conscientiously followed, the ideal results in monocular esotropia can be obtained.

COLOBOMA LENTIS WITH ABNORMAL CILIARY BODY

BY

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BIRMINGHAM

THE following case is of interest, not only from its rarity, but because had its real nature not been recognized a harmless anomaly might have lead to the removal of a useful eye.

Mrs. D., aged 23 years, came to the Eye Department of the Coventry Hospital saying that she thought she needed glasses.

My house surgeon, Mr. Logan, examined her and found in the right eye what he thought might be a tumour in the ciliary region.

The following conditions were present:—

Externally the eye, which has a brown iris, is perfectly normal in aspect, without a trace of injection. The pupil is normal in size and reactions. The media are clear and the fundus normal. The vision of the right eye is 6/12 that of the left, 6/4·5. There is no material error of refraction. The tension of each eye is normal.

Examination with a + 20 D. lens behind the ophthalmoscope shows in the lower aspect of the vitreous chamber, and close behind the lens, a smooth grey circumscribed mass suggesting a sarcoma of the ciliary body.

Focal examination with the ordinary loupe by the light of a half-watt 60 c.p. bulb reveals a brown mass, sponge-like in texture, with no trace of irregularity or of vascularization. In its centre but apparently unconnected with it, is a yellowish area. When the

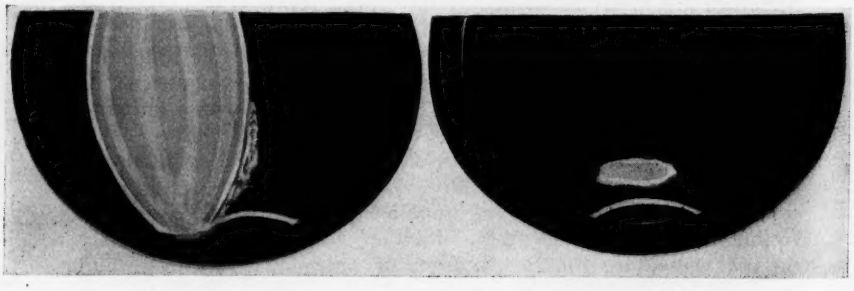

FIG. 1.

The eye seen in optical section. The coloboma of the lens is clearly seen. The flocculent deposit is shown on the back of the lens. The brown mass occupies about one fifth of the circumference of the pupil.

FIG. 2.

The interior of the eye seen by focal illumination. The coloboma is seen below with its usual shining edge. The brown mass with the yellow area occupies the lower third of the picture.

pupil was dilated a small coloboma of the lens was discovered. There appeared to be a small opacity in the lower part of the lens.

The slit-lamp examination with the broad beam showed that the mass had the texture and colour of ordinary uveal tissue, and that there was no vascularization. It merged with the ciliary body without any abrupt edge, and had no irregularity of surface that might be present in a new growth.

The vitreous was perfectly normal showing the usual films without any trace of infiltration with cells or larger particles.

When examined with the narrow beam in optical section, it was seen that the white area seen by focal illumination on the surface of the mass was really a flocculent filamentous deposit on the back of the lens. It extended downwards and was lost in the region of the coloboma.

The lens itself, except in the colobomatous area was perfectly clear, and showed the usual sutures and zones of discontinuity.

I came to the conclusion that the mass was due to an abnormal development of the ciliary body in the lower sector of the eye, probably a fusion of several processes, and that there could be no question of sarcomatous growth. Apart from the fact that the lens was obviously implicated in the abnormal picture, the absence of vascularization, the normal aspect of the vitreous, and the normal tension as estimated with the Schiötz tonometer, were evidence against tumour formation.

Mrs. D. has now been under observation for six months and no change has taken place in the eye.

CONSERVATION OF EYEDROPS

BY

DR. KLEIN MIKLÓS

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THE sterility of eyedrops is very important because they are used after operations which open the globe and also after perforating injuries, when it is possible for micro-organisms to be carried with the drops into the internal part of the bulb, and thus cause dangerous infections.

First, we had to examine whether the eyedrops are infected or not. It is well known that most eyedrops, chiefly the alkaloids, become after more or less time opalescent, and in these there

appear white filaments. When we inoculate from such a fluid an agar-agar plate, after 10-12 hours we find it amply furred with different colonies of micro-organisms.

Apart from the bactericidal remedies we succeeded in breeding micro-organisms from all the eyedrops in general use: atropine, scopolamine, eserine, pilocarpine, holocaine, homatropine, and zinc-sulphate.

The cultures grew bac. myentericus, bac. prodigiosus, and other non-determined chromogen saprophytes; also *staphylococcus albus and citreus*. Besides these we found some kinds of fungi the identification of which is now in progress.

The infection of the eyedrops occurs by touching the patient's face or conjunctiva with the glass dropper. The air infection also plays a part. So we presumed that the bacterial infection varies in different countries and therefore we gave up the exact identification. It is important that we found in the infected eyedrops staphylococcus which can cause eye infections.

We determined the duration of sterility of eyedrops. When we opened the bottle several times daily, *i.e.*, only air infection, we find almost all fresh, sterile prepared eyedrops infected before 48 hours. Of course, in these cases the liquid seemed perfectly clear and only on nutrient media we proved the presence of infection. Consequently, the infection of eyedrops is not recognized by the naked eye.

From the above it is evident that the conservation of eyedrops is very important. For this we have many possibilities. The daily sterilizing is very tiresome and the boiling of the easily decomposable alkaloids spoils them. It is recommended also to add 1 : 10,000 mercury-oxycyanate to the eyedrops. We object to this practice. Many conjunctival sacs do not tolerate this treatment and the mercury salt has a reaction against the alkaloids and can decompose them.

The cover over the bottle does not protect against the air infection because when the drops are used the glass is open.

The conservating substances used in the laboratory such as phenol, salicylic acid, benzoic acid, and thymol cannot be used, partly because of the caustic effect, and partly because of their disagreeable odour. A long time ago the conservating effects of resin benzoic were discovered. We knew too that its effect does not depend on the content of benzoic acid, because the resin, probably due to its ester content, conserves better. Sabalitschka drew attention to the ester of the benzoic acid, which has a greater advantage in comparison with the other conservating substances. They are neutral, have no smell, and do not dye. They are soluble with difficulty in water. We know that the dividing co-efficient lipoid: water also has a rôle in the process of disinfection. The

following figures taken out of Sabalitschka's work show that the dividing co-efficient becomes a high value in the different esters. This table also shows the hindering and destroying effects on cultures of the staphylococcus. All the comparisons are made to phenol, the value of which is taken as 1.

TABLE I.—SABALITSCHKA

	Staphylococcus destroyed.	Staphylococcus growth hindered.	Lipoid : water div. co-eff.
Phenol - - - -	1	1	1
Methyl p. oxybenzoat. -	2·6	3	1·2
Ethyl p. „ -	7·1	8	3·4
Propl. p. „ -	15·0	17·0	13·0

The small quantities which are carried with the eyedrops into the culture are from toxic point of view not taken in account. Schübel and Manger established by experiments on dogs and rabbits, that per os 2 gr. of the methylester for every kg. body weight is toxic, and 3 gr. per kg. body weight is lethal; from the ethylester 4 or 5 gr. per kg. body weight is toxic or lethal; from the propylester 3-4 or 6 gr. per kg. body weight toxic or lethal. Accumulation does not follow. The esters are less toxic than the para-oxybenzoic acid itself.

Since the infected eyedrops may contain bacteria which have a rôle in the pathology of the eye, it seemed important to find out a method of conservation for eyedrops so that their effect remains unchanged. We should conserve eyedrops because they contain expensive alkaloids and at large hospitals economy is important. Finally, by means of conservation the physician is released from the duties of controlling the sterility of eyedrops.

We tried for eyedrop conservation the use of the esters of benzoic acid because as we mentioned above they have many advantages.

In our researches to conservate eyedrops, we experimented with the three esters and their compounds which are soluble in water. The method was the following. We added the above-mentioned substances to agar-agar in different proportions. The nutrient media thus prepared, we inoculated with micro-organisms taken from infected eyedrops. In order to put in all nutrient media an equal quantity of micro-organisms we made first a uniform emulsion in the mortar and we placed the micro-organisms in all test tubes with the same platinum hook. Besides this we made our investigations with fluid nutrient media and with blood-agar plates. The

micro-organisms which we used were derived from different eye-drops and from an orbito-phlegmon pus, consisting of a mixed culture of streptococci, staphylococci, and bacillus coli.

The results are as follows:—

TABLE II

*Methyl p. oxybenzoate	0·1%	0·2%	Control	
after 24 hours	+ + +	–	+ + + +	
,, 48 ,,	+ + + +	–	+ + + +	
,, 72 ,,	+ + + +	–	+ + + +	
*Methyl p. oxybenzoate natr.	0·1%	0·2%	Control	
after 24 hours	+ + + +	+	+ + + +	
,, 48 ,,	+ + + +	+ +	+ + + +	
*Ethyl p. oxybenzoate	0·075%	0·1%	0·15%	Control
after 24 hours	+ + + +	+	–	+ + + +
,, 48 ,,		+	–	+ + + +
,, 72 ,,		+ +	–	+ + + +
*Ethyl p. oxybenzoate natr.	0·1%	0·15%	0·2%	Control
after 24 hours	+ +	±	–	+ + + +
,, 48 ,,	+ + +	+ +	–	+ + + +
,, 72 ,,	+ + +	+ +	–	+ + + +
*Propyl p. oxybenzoate	0·03%	0·04%	0·05%	Control
after 24 hours	+ +	±	–	+ + + +
,, 48 ,,	+ + +	+	–	+ + + +
,, 72 ,,	+ + +	+	–	+ + + +
*Propyl p. oxybenzoate natr.	0·05%	0·06%	0·075%	Control
after 24 hours	+ +	–	–	+ + + +
,, 48 ,,	+ + +	–	–	+ + + +
,, 72 ,,	+ + +	+	–	+ + + +

The results on fluid nutrient media are a little better than those on solid ones. To control the growths on a fluid medium is not so easy and it is far better to compare on a solid medium, especially when there are only one or two colonies. Therefore, the majority of examinations were done on solid media. The figures do not show the average, but the worst values which occurred. In the propyl esters case the micro-organisms were already hindered in growing by 0·04 per cent. and with the natr. compound by 0·05 per cent. concentration. Only in two cases we found a tiny growth at the concentration mentioned above: in one case by mixed strepto-staphylococcus inoculation. Of course, the hindering effect depends also on the virulence and not only on the concentration of the disinfectant applied.

We found that all three blends are able to conserve. In the case of methylester the effective concentration is at 0·2 per cent.; ethyl-ester 0·15; and propylester 0·05 per cent.; with the natr.

* Manufacturer: Ful. Penner, Berlin-Schöneberg.

compounds which are soluble in water the effecting concentration is about 1/5 more. It seems reasonable to use the propyl p.oxybenzoate for the conservation of eyedrops because the necessary concentration is with this substance the least, and its toxic effect is also the smallest.

The micro-organisms which are smeared on agar plates and are hindered in growing, die after some time. Our results concerning this are as follows: Micro-organisms cultured on prepared agar plates die in 6-10 days. The method was as follows. With the above-mentioned substances prepared agar-tubes were inoculated and the tubes were washed at various times with sterile bouillon and this bouillon was placed in a thermostat. By applying fluid nutrient media the method was simpler because at various times we inoculated a drop into sterile bouillon. In the fluid medium the staphylococcus perishes at the concentration 0·1 per cent. propyl p.oxybenzoate natr. in less than 48 hours.

We do not forget that during the investigations the micro-organisms are on a nutrient medium, *i.e.*, among favourable life conditions and as they are only on the surface of the agar plate they cannot be in contact with the disinfectant substance. In the eyedrops the conditions are worse, because they do not have the best medium for growth for in the fluid they are in permanent and direct contact with the disinfectant.

At the practical application we found that the water soluble natr. compound of the propyl ester gives with the holocaine and zinc-sulphate an opalescent solution, and the eserine becomes red in a few hours. Therefore, we cannot apply it. The pure ester is good for the purpose of conserving eyedrops. It is advisable to prepare first an alcohol stock solution 1 : 4 because the pure ester is very difficult to dissolve in water. The dilution we make from this stock solution. In preparing the necessary solution, the water must be kept in motion and the stock solution must be added only in drops. If it precipitates the whole must be boiled, and the solution remains clear after cooling. The boiling is good because by means of it the small quantity of alcohol of the stock solution evaporates. The water thus prepared is ready for eyedrops.

The propyl p.oxybenzoate we applied in 0·05 per cent. concentration. The eyedrops thus prepared are durable and they remain sterile. All substances used in eye treatment are easily soluble with a 0·05 per cent. propyl p.oxybenzoate solution. A further advantage is that the eserine does not become red, even after a long time.

First we tried on rabbits' eyes and later on patients. We can determine that with the above-mentioned substance, prepared eyedrops do not irritate the conjunctiva.

Conclusions

We tried different esters of the para-oxybenzoic acid in order to conserve eyedrops and found that the most suitable of these is the propylester in 0·05 per cent. concentration. The eyedrops thus prepared hinder entirely the growth of the micro-organisms and by the mild bactericidal effect those which enter the eyedrops die.

The eyes tolerate these prepared eyedrops well.

This method of conservation is recommended, because by it the physician does not have trouble in keeping the eyedrops pure.

Also from an economical point of view it is necessary to conserve eyedrops, because many of the alkaloids which are used in it are expensive.

LITERATURE

Th. Sabalitschka.—*Arch. de Pharmacie*, Vol. XXIX, p. 272, 1929.

ANNOTATION

International Illumination Congress, 1931

During September, there was held in Great Britain an International Illumination Congress. This Congress forms an adjunct to the meetings of the International Commission on Illumination. This latter body was originally founded in 1900 as the International Commission on Photometry and was changed in 1913 to its present form. It is in essence a co-ordinating body, being composed of delegates from National Illumination Committees which exist in most civilized countries. Recently, the meetings of this Commission have been preceded by a Congress to which everybody interested in illumination could come.

This particular Congress has attained some notoriety owing to the flood-lighting, in honour of the Congress, of some of the public buildings in London and elsewhere. It is not necessary to say very much about this feature. Some of the illumination was merely a glare. Occasionally, it rendered visible architectural features which are not ordinarily observed and the flood-lighting of some of the flower beds in St. James's Park was interesting, as showing what can be done. It is only fair to say that no charge fell upon the public authorities for all this expenditure of energy.

The Congress assembled in London on September 1, and on the 3rd began a tour through the principal industrial centres of Britain,

going first to Glasgow for three days and then to Edinburgh, Sheffield and Birmingham. At each place meetings were held, papers were read and discussed, and local works, etc., inspected. The last meeting place was Cambridge, where the International Illumination Congress held its meeting, so that those who were interested could follow on. This concluded on September 18th and the whole Congress returned to London on September 19th in time for the Faraday Centenary Celebrations, which began on September 21st, and then on September 23rd came the meeting of the British Association for the Advancement of Science.

The reason for these peregrinations was to give the delegates a general idea of the great industrial centres of Britain and also to stimulate local interest in lighting.

More than 150 papers on all branches of the subject were brought before the Congress, ranging from the recent developments of lighting of the factory, house, shop, theatre, church, to aviation and architectural lighting, coloured signal glasses, motor car headlights and luminous traffic signals, etc., a very varied programme containing a vast amount of useful information, but we regret to notice that the physiological aspect of illumination has been almost entirely neglected. There are only five papers dealing with anything even remotely connected with the physiology of vision and yet this aspect of the problem is fundamental. It is within our recollection that the late Mr. Leon Gaster, the founder of the Illuminating Engineering Society, always emphasized the importance of the human element, and we feel certain that if he had been alive it would not have been thus neglected. It is to be hoped that in future Congresses this question will receive the consideration it deserves and that the teaching and example of Mr. Leon Gaster will be followed.

It is useless to attempt to summarize the papers read at this Congress, and even a mere list would occupy too much space, but probably ophthalmic surgeons would be most interested in those dealing with photometry. It is noteworthy that much progress has been made in measuring the intensity of coloured light and in improving the accuracy of photometers.

Among the physiological papers there are two on visual acuity under varying illumination and one on a new apparatus for testing visual function.

XIVth INTERNATIONAL OPHTHALMIC CONGRESS

Communication from the International Ophthalmological Council

MADRID CONGRESS, 1933

I. The XIVth International Ophthalmological Congress will take place in Madrid at the beginning of April, 1933. The principal subjects to be discussed at this congress, are

(*a*) *Tuberculosis of the Iris and Ciliary Body.*

Reporters	*Address*	*Subdivision of subject*
Dr. E. V. L. Brown -	University of Chicago, 950 E. 59th Street, Chicago III. (U.S.A.)	Modern methods of treatment
Prof. Dr. J. Igersheimer	Brentanostrasse 1, Frankfort a. Main, Germany	Pathological anatomy
Dr. Henri Lagrange -	5, Avenue Daniël Lesueur, Paris, VII, France	Diagnosis and differential diagnosis

(*b*) *Detachment of Retina.*

Dr. H. Arruga - - -	Aragón 271, Barcelona, Spain	Aetiology
Prof. Dr. G. Ovio - -	Viale Castro Pretorio 66, Rome, Italy	Medical treatment
Prof. Dr. A. Vogt - -	Rämistrasse 73, Zürich, Switzerland	Operative treatment

Authors of papers connected in any way with one of these two subjects are requested to send their papers to the reporters in question.

II. All communications to the congress shall refer essentially to one or other of the above-named two subjects. Two or three meetings, however, will be reserved for free communications, including one demonstration meeting.

The number of communications shall be limited, both as regards those that deal with the principal subjects and also the free communications. For this reason papers will have to be in the hands

of the secretary of the Internat. Ophthalm. Council on *December* 1, 1932, *at the latest.*

In order to secure the best selection from all papers sent in, an international committee of ophthalmologists shall previously examine all communications. After that, authors of communications will be informed if their papers are to be accepted for reading or not.

III. With regard to reports, drawn up for the XIIIth Internat. Ophthalm. Congress at the request of the Internat. Ophthalm. Council, the complete commission of reporters shall take their final decisions during the XIVth Congress. These decisions shall be communicated to ophthalmologists individually, to ophthalmological associations and, if necessary, to the governments with a view to their practical application. Consequently, all ophthalmologists who wish to make any observations on the conclusions in the above-named reports (to be found in the *Proceedings of the XIIIth Congress*, Vol. IV) are earnestly requested to address observations or comments before May 1, 1932, to the secretary of the Internat. Ophthalm. Council.

IV. The reporter on the subject "Pathological anatomy of tuberculous iridocyclitis," Prof. Dr. J. Igersheimer, requests all those who possess special preparations or sections of cases of tuberculous iridocyclitis, to forward these to his address for examination.

V. Oculists who wish to widen their professional knowledge abroad, either by a short visit to a foreign clinic, or by way of a longer stay as a volunteer, may apply to the secretary of the Internat. Ophthalm. Council, who will try to find clinics to suit them.

VI. In the year 1929, during the XIIIth Internat. Ophthalm. Congress, at Scheveningen, the Council decided to start an ophthalmological library. Most of the ophthalmological periodicals have already been collected at Leiden, where the library is established. The object is to collect there everything issued in the domain of ophthalmology. Our colleagues are therefore requested to address a reprint or copy of their publications (viz., reviews in periodicals, books, theses, etc.) to the librarian of the Internat. Ophthalm. Library, Dr. J. Kroon, Stationsweg 25, Leiden, Holland. Further particulars on the working method of this library will follow in due course.

Address: Inrichting voor Ooglijders,
Oostmolenwerf 5,
Rotterdam, Holland. *Secretary:* E. MARX.

ABSTRACTS

I.—TRACHOMA

(1) Finoff, William C., Thygeson, Phillips (Denver).—Bacterium granulosis in trachoma. *Arch. of Ophthal.*, April, 1931.

(1) **Finoff** and **Thygeson** give the results of their experimental work in a long and interesting paper. Their conclusions are as follows:—

(1) A minute Gram-negative motile rod, apparently identical with that described by Noguchi under the name of Bacterium granulosis, has been recovered from five of thirteen white persons, and from one Japanese with advanced trachoma. The objections raised by Lindner regarding the original Indian cases studied by Noguchi cannot be applied to our cases because of the presence of pannus and advanced scarring in all but two cases. Bacterium granulosis was recovered also from two of fourteen trachomatous Indian children studied. (2) The bacterium is either rare or absent in non-trachomatous diseases of the conjunctiva. (3) Bacterium granulosis may be considered more characteristic of trachoma than the inclusion bodies of Prowazek or the initial bodies of Lindner, since it can be recovered from patients having advanced disease at a time when the inclusion bodies and initial bodies are usually absent. (4) The probable reason for the failure of former investigators to discover the organism lay in their use of unsuitable media and their failure to cultivate at the optimum of 30° C. (5) The bacterium was not agglutinated by serum that was tried from a single case of trachoma. (6) Skin tests with Bacterium granulosis antigen have been uniformly negative, with one exception. (7) Bacteria morphologically identical with Bacterium granulosis have been seen on and in the epithelial cells in smears taken from trachomatous patients and from animals having the experimental disease. (8) Inoculation of *Macacus rhesus* monkeys with suspension of Bacterium granulosis has resulted in the production of a granular conjunctivitis identical with that described by Noguchi and also with that resulting from the injection of human trachomatous material. (9) Contact infection occurred in one of two monkeys exposed. (10) The bacterium has been successfully recovered from two animals having an advanced disease. (11) Our results would seem to confirm those of Noguchi in all essential respects. (12) Because of the fact that trachoma in monkeys is not identical with that in human beings, it may be necessary to resort to human inoculations to prove or disprove conclusively the aetiological relationship of Bacterium granulosis to trachoma.

One interesting point is that Bacterium granulosis is sensitive to cocaine in quite low concentrations, though it is resistant to

procaine hydrochloride in higher concentrations. This fact may account for the failure of some observers to isolate the organism from cases of trachoma in human beings.

F. A. W-N.

(2) **Cucco (Palermo).—The present state of the problem of trachoma. (Lo Stato Attuale del Problema Etiopatogenetico del Tracoma).** *Ann. di Ottal. e Clin. Ocul.*, December, 1930.

(2) The hope that was raised two years ago, that this problem was to be solved by the discovery of Noguchi, has not been fulfilled. In this paper, **Cucco** reviews the various communications which have appeared since 1928, and concludes that Noguchi's bacillus is not the specific cause of the disease; he bases this opinion chiefly on the fact that the conjunctivitis following inoculation has never given rise to those cicatricial changes which are characteristic of true trachoma, but has been followed often by spontaneous cure without any trace of scarring.

HAROLD GRIMSDALE.

(3) **Addario (Cantania).—The aetiology of trachoma and the new views of diagnosis treatment, and prophylaxis, which arise therefrom. (L'Etiologia del Tracoma ed i Nuovi Criteri Diagnostici, Terapeutici e Profilattici che ne Derivano).** *Ann. di Ottal.*, December, 1930.

(3) **Addario** takes a very different view (*cf.* No. 2, supra); for him, Noguchi has proved the Bacterium granulosis to be the true cause of trachoma. Addario has infected a blind eye, with the patient's consent, with a culture of Bacterium granulosis sent from New York, and has watched the development of true trachoma. This is an observation of great importance, and one that must have weight in forming judgment.

Addario lays stress on the double process involved in trachoma, the one neoplastic, the formation of the granules, and the other inflammatory; from the interaction of these two processes springs the polymorphism of the disease. He points out that in the early stages, the disease gives rise to few urgent symptoms; the sufferers then, when adult, feel no necessity to stay away from their work, and grudge the time necessary to attend at clinics for treatment; it is essential, therefore, that any form of treatment shall be simple, and possible to use at home without too great inconvenience; Addario recommends an ointment of 10 per cent. copper in vaseline, to be used every evening after return from work. In schools the daily treatment may be carried out for the children by a nurse. If trachoma is treated efficiently in the early stages, all corneal complications will be averted and there will seldom be necessity for operative interference on the lids.

HAROLD GRIMSDALE.

(4) **Sjögren, H. (Stockholm).—Cold in the treatment of trachoma. (Ueber Kältebehandlung des Trachoms).** *Acta Ophthal.*, Vol. VIII, p. 296, 1930.

(4) The conflicting opinions held on the method of treating trachoma by cold is recalled by Sjögren who strongly supports it, as a result of his experience with 42 cases. In the author's opinion the technique employed explains the divergent views. For successful results he advocates the application of a pencil of CO_2 snow for 15-20 seconds: each part of the tarsal conjunctiva and fornix to have the pencil firmly applied for this period. Until it has thawed the lid should not be allowed to come in contact with the cornea, otherwise pain is experienced. In the subsequent oedema or even formation of pseudo-membranes the superficial follicles become extruded and the deeper ones absorbed. The usual after-treatment with perchloride and copper should be continued after the subsidence of the reaction. The application of CO_2 snow may be repeated after 7-10 days. In no case did the author observe scar-formation on the conjunctiva, but the cases were not followed up long enough: although no scars are formed the follicles do not re-appear.

ARNOLD SORSBY.

(5) **Roche, Charles (Marseilles).—The contagion of trachoma. (A clinical study).** (**La Contagion du Trachome. [Etude clinique]**). *Arch. d'Ophtal.*, October, 1930.

(5) The writer of this paper has practised for many years in an area in which trachoma is common. Since 1924 this disease has been listed as notifiable in France and measures designed for its control and eradication are being pursued. **Roche** after remarking that " whatever administrative measures we may adopt we cannot obtain complete control of trachoma (as indeed of any contagious disease) until we possess exact knowledge of the modality of its contagion," proceeds to discuss some of the perplexities concerning the propagation of this disease and the various explanations which have been offered. He submits four propositions based upon observation in his own practice and appends clinical evidence in support of each one.

(1) Isolated cases of trachoma occur in localities in which the disease is unknown.

(2) In large families dwelling in insanitary homes a single member of the household is affected.

(3) Cases occur, and are not very exceptional, in which the disease is strictly unilateral.

(4) Persons who for years are in daily contact with trachoma remain free from the disease.

The most surprising of these observations, in the author's

opinion is number 2, in which perhaps the mother of a large poor family is attacked, while the father and all the children escape. It seems unlikely that such a statement would be true of any other contagious disease, and it is not surprising that such observations have originated an idea that trachoma is not contagious. Its contagiousness, however, has been established beyond question.

The contagion of trachoma may appear to us mysterious or at least capricious, in its behaviour, and it is not easy to explain some of the apparent contradictions often observed. Various hypotheses have been offered to account for its vagaries. The author discusses several and rejects them all, though he suggests that the hypothesis which best explains the observed facts is that which assumes the existence of an intermediate host such as has been demonstrated in malaria and other diseases. His own theory, which is " contrary to universally accepted ideas " is that the period of contagiousness of trachoma is of short duration, perhaps anterior to the appearance of the granulations. This hypothesis he thinks may throw light upon some of the problems concerning the spread of trachoma, such as the difficulty in discovering the pathogenic agent, the variation and uncertainty in the results of experimental transmission, etc. Moreover, it is in no way adverse to the existence of a specific microbe, or of its persistence in the conjunctiva; a microbe which loses its virulence rapidly, or which has need of an associate, of ephemeral character, in order to be transmissible.

J. B. LAWFORD.

II.—RETINA

(1) **Luntz, G. (Vienna).—The effect of cautery puncture on the animal eye. (Ueber die Wirkung der perforierenden Kauterisation am Tierauge).** *Zeitschr. f. Augenheilk.*, Vol. LXXIII, p. 380, 1931.

(1) **Luntz** has performed cautery puncture on the eyes of six rabbits and one monkey. A cautery of 1 mm. point was used, and the choroid perforated with the cold point.

In five eyes removed five to seven days after the operation, the track was found to be filled with vitreous or blood; no granulation tissue was present. In two eyes removed after 22 days there was an ingrowth of episcleral tissue in the track. The sclera was necrotic for an area of 0·5-1 mm. around the hole; the choroid was destroyed for a similar area but the retina was reduced to a thin membrane for an area of 3·5 mm. No other results besides these purely local ones were observed.

No difference could be detected in the results, whether the cautery

employed was red-hot or glowing white. Likewise it made no difference whether it was applied for two seconds or for 20.

A point of significance is the relatively extensive destruction of the retina, for this may explain why fresh holes are formed at the site of the puncture.

ARNOLD SORSBY.

(2) **Amsler, Marc (Lausanne).—Anatomical and clinical notes on cautery puncture. (Remarques cliniques et anatomiques sur la thermocauterisation perforante).** *Ann. d'Ocul.*, Vol. CLXVI, p. 871, November, 1929.

(2) **Amsler** describes a case of iritis with glaucoma in an eye almost blind, in which he performed experimental cautery puncture by Gonin's methed fifteen days before enucleation. It had been decided to remove the eye on account of pain. The tension had remained at about 40 mm. mercury by Schiötz tonometer for some days, but the day following the operation the tension rose to 60 mm. after the usual extreme fall due to the puncture. The tension fell gradually in three days to below 30 mm. and then to about 12-15 mm. until the eye was excised. Amsler regards the occurrence of low ocular tension after cautery puncture as a powerful argument against the theory of pathogenesis of retinal detachment supported by certain ophthalmologists, in which the detachment is regarded as being due to the low tension.

The main points of interest emerging from the examination of this eye are (1) the existence of a thin thread or band in continuity with the vitreous (fixed in formalin) holding the retina in contact with the cicatrix, and (2) the extent of the cicatricial vascular granulation tissue on the inner surface of the sclerotic at the site of puncture—3·5 × 5 mm.

HUMPHREY NEAME.

(3) **Rubbrecht, M. R. (Bruges).—The closure of retinal tears. (L'Obturation des déchirures de la rétine).** *Bull. de la Soc. belge d'Ophtal.*, November, 1929.

(3) The communication deals with questions of technique in treatment rather than of pathology.

The position of the retinal tears is discussed, and then the most important question of localisation, the determination of the degree of longitude and latitude. A sufficiently accurate marking of the longitude is not difficult and **Rubbrecht** shows by diagrams how he deals with this, but the exact definition of the latitude is more difficult, but according to the writer is not necessary if his method of cauterization is employed.

Retrobulbar injection is employed and is useful for the slight exophthamos as well as for the anaesthesia.

The internal or external rectus is resected, and the stump gives a good purchase for rotation of the eye. The conjunctival incision is then continued parallel to the limbus in the required direction, and the sclerotic can be exposed as far back as is needed by means of a retractor. The meridian of longitude is then marked out with Indian ink along the guiding thread in the usual way, and the distance previously estimated, measured out from the limbus and marked. The scleral incision is made on either side of this mark, 4 mm. in length, and this tends to neutralize the error which must be present in the measurement of the meridian of latitude.

The sclerotic is divided slowly and carefully in the whole 4 mm., and when the dark choroid appears this is incised and the subretinal fluid escapes. A fine cautery is then passed along the whole length of the incision.

Rubbrecht uses the galvano-cautery and deprecates the plunging of the point into the eye.

The divided muscle is then sutured, and the patient remains in bed for eight days only.

O. GAYER MORGAN.

(4) **Amsler, M. (Lausanne).—Markers for cautery puncture. (Marqueurs pour la thermo-ponction oblitérante de Gonin).** *Ann. d'Ocul.*, Vol. CLXVII, p. 115, 1930.

(4) **Amsler** has designed a series of markers for indicating and marking with Chinese ink the point on the sclerotic corresponding with the retinal hole at which cautery puncture has to be made in cases of detachment of the retina. A battery of thirteen markers has been made. Each consists of a handle, a corneal portion to overlie the cornea, a curved scleral portion to overlie the sclera and a beak at right angles to the curved portion. The beak is of 2·5 mm. length and is grooved to hold the ink. The scleral portion varies in the different markers from 10 to 22 mm. in length to suit every case. The whole instrument is of rustless steel (M. N. Dugast, 108, Boulevard St. Germain, Paris). The illustrations indicate clearly the properties and method of using the markers.

HUMPHREY NEAME.

(5) **Hamilton, J. Bruce (Hobart).—The Gonin operation for the cure of idiopathic retinal detachment.** *Med. Jl. of Australia*, May 16, 1931.

(5) **Hamilton** at the beginning of this paper briefly reviews some of the events and discussions which have led to the adoption of Gonin's operation for the cure of idiopathic detachment of the retina. He was senior house surgeon at the Moorfields Eye Hospital when the first sixty patients suffering from detachment of the retina were treated by Gonin's method and it is mainly

from this wealth of clinical material that he has made his observations.

He describes the methods of localizing retinal apertures and of marking these on the exterior of the globe; the types of hole encountered, and a reflection on those that he considers suitable or unsuitable for this operation.

In referring to a hole at the macula he states that this is "a position quite inaccessible."

The operative technique and post-operative treatment are well described, and much valuable information of a practical nature is given. This paper is well worth reading in the original. It is obvious that Hamilton has had much experience of this operation and has made some sound observations about its technique and prognosis.

H. B. STALLARD.

(6) **Redslob, E. (Strasbourg).—Neuro-epithelial proliferation in detachment of the retina. (Hyperplasie neuroépithéliale dans un décollement de la rétine).** *Ann. d'Ocul.*, Vol. CLXVII, p. 104, 1930.

(6) **Redslob** describes in detail the results of his examination of an eye of a child, aged five years, that had suffered from an acute illness at the age of two years. The child had shown evidence of a meningitis with fever, also albuminuria and a purpuric eruption of the skin. In the course of the illness endophthalmitis developed in one eye, and a secondary cataract supervened. At the age of five years the eye was excised for glaucoma.

The feature of special interest was the hyperplasia of neuro-epithelium in the ciliary region. The retina was found to be detached. In the pars plana ciliaris the inner layer of ciliary epithelium had proliferated to a remarkable extent. Two especial forms of modified cells were found in addition to considerable neuroglial proliferation. There were cylindrical cells arranged in tubule formation with a definite lumen and also greatly elongated cells with long protoplasmic fibres anastomosing with one another. Redslob compares these cells (1) with the cylindrical cells which line the primitive neural cavities and (2) with spongioblasts which in a later stage of development form a framework of anastomosing fibres and extend from the inner surface of the primitive neural cavity into the deeper layers of cells. These cells as a result, it is assumed, of inflammation have undergone proliferation in the direction of taking up the functions of primitive neural cells. In none of this proliferation was there the slightest evidence of malignant growth. Redslob compares his case with one reported by Fuchs of a similar nature but in which the cells showed evidence of malignant growth. He makes the suggestion that not only

malignant growths such as this affecting the ciliary neuro-epithelium but also the less rare neuro-epithelioma (*syn.* glioma retinae) may perhaps be the result of a previous inflammation occurring in intra-uterine life.

HUMPHREY NEAME.

(7) **von Pelláthy, B. (Budapest).—Retinal detachment and congenital uveitis in two sisters and a brother. (Ablatio Retinae und Uveitis congenita bei drei Geschwistern).** *Zeitschr. f. Augenheilk.*, Vol. CXXIII, p. 249, 1931.

(7) **Pelláthy** records the findings in three children, two sisters and a brother, who became blind within the first 18 months of life. There was no consanguinity amongst the parents, whose history is quite negative. There were no other children. In the two elder children, aged 6 years and 3 years respectively, the eyes were in an advanced stage of shrinking. Dense vitreous opacities precluded a good view of the fundus, though retinal detachment could be diagnosed. In the youngest, aged 6 months, serous retinal detachment could easily be seen; vitreous opacities and choroidal lesions were likewise observed. The children were mentally and physically backwards. No history of stigmata of degeneration in the family could be obtained. The author sees in foetal uveitis the cause of the ocular degeneration, and recalls similar cases recorded in the literature.

ARNOLD SORSBY.

(8) **Guist, G. (Vienna).—A new operation for detachment. (Eine neue Ablatio-operation).** *Zeitschr. f. Augenheilk.*, Vol. LXXIV, p. 232, 1931.

(8) The good results obtained at Lindner's clinic by Gonin's operation for detachment have not been maintained, for recurrences have often taken place owing to fresh hole formation at the site of the cautery scar. Furthermore vitreous haemorrhage induced by the reactionary vascularity at the site of the cautery scar produces a vicious circle if further operation is necessary. These difficulties led **Guist** to search for a less drastic procedure than thermo-cautery. From experiments on rabbits he found cauterization with a caustic-potash stick much milder and safer, sufficient reaction being produced to cause an adhesive exudate to seal the hole with but little destruction of tissue; there is little danger of vitreous haemorrhage.

The main points in the technique are:

(1) Having localized the hole, the corresponding area on the sclera is trephined (after a conjunctival flap has been dissected up).

(2) Haemorrhage at the trephine hole having been controlled a paraffin-mounted caustic-potash stick freshly sharpened (this

being necessary owing to its hygroscopic nature) is introduced into the hole for one or two seconds. The choroidal area thus treated is immediately counter-treated with 0·5 per cent. acetic acid applied by means of a moistened probe.

(3) The choroid is then perforated with a blunt probe, thus establishing contact with the subretinal space, the retina itself not being touched.

(4) If a large hole has to be sealed, one or two trephine areas at the margin of the hole must be thus fully treated, though some more trephine holes may have to be cauterized (without actual perforation of the choroid).

ARNOLD SORSBY.

BOOK NOTICES

Seeing : a Partnership of Lighting and Vision. By M. LUCKIESH, D.Sc., and FRANK K. MOSS, E.E. Pp. 241. London : Bailliére, Tindall & Cox. 1931. Price, 22/6.

The authors of this book are physicists engaged in the research laboratory of the General Electric Company of America, at Nela Park, Cleveland. Much of the most valuable research work on the problems of lighting have emanated from this laboratory in recent years. So far as the physical problems of lighting and vision are concerned the authors are thoroughly reliable. They have not neglected the more outstanding physiological and psychological problems, but on these they write with less authority. The book is a valuable compendium of miscellaneous information dealing with visual acuity, contrast, brightness, adaptation, glare, fatigue, and so on. Some parts are very elementary and others are platitudinous. The most valuable parts require a considerable knowledge of physics and can only be thoroughly grasped by those having facility in the reading of graphs.

Dr. Luckiesh is a prolific writer on these subjects—16 books published between 1915 and 1930 are advertised in this volume. The bibliography in this book consists of 48 items, all of which are American with the exception of two (König and Helmholtz).

Das Werk von Professor Dr. Otto Haab. By Dr. JACOB STREIFF. Basel: Benno Schwabe & Co. 1931. Price, 2·5 Swiss francs.

This tribute to Professor Haab, "in his honour and in commemoration of his 80th birthday," is from the pen of a former pupil : it has been conceived in a spirit of sincere admiration for a greatly respected ophthalmologist and teacher, and will be read with appreciation by many in the world of ophthalmology who have no personal acquaintance with the Professor.

Streiff's brochure contains the record of a long professional career of unremitting activity in clinical and experimental investigation, carried on coincidently with practice and teaching. He recounts, in roughly chronological order, Haab's very large literary output, and notes that his books and shorter contributions are fairly representative of the history of ophthalmological progress during the last half-century.

Naturally, a prominent place is given to the introduction of the large electro-magnet for the removal of intra-ocular foreign bodies. This beneficient discovery (which, in Haab's words, he came upon by " ein glücklicher Zufall ") will remain a permanent memorial to the Professor when much of his other work has been forgotten. Another enduring memorial is the fine "Universitäts Augenklinik" in Zürich, planned by Haab and erected under his personal supervision. When opened in 1896, it was one of the most complete institutions of its kind, and was at once the pride and delight of the Professor. Streiff mentions, as among the noteworthy features of Haab's professional work, his keen interest in systemic diseases such as tuberculosis, congenital syphilis, circulatory disorders, etc. which closely concern the ophthalmologist, his constant endeavour to perfect the treatment of wounds (in his special territory) and his ever present aim of widening the bounds of ophthalmological knowledge. As a teacher he gave no encouragement to the discussion of theories, but strove to incite habits of accurate observation and the acquirement of sound knowledge.

A pleasing portrait of Professor Haab, and a photo of a "magnet-operation " in which he is the manipulator, add interest to this attractive story.

Detachment of the Retina: a contribution to the Study of its Causation and Treatment. By J. RINGLAND ANDERSON, Ophthalmic Surgeon to the Alfred Hospital, Melbourne, with a foreword by Sir John Herbert Parsons. Cambridge University Press. Published for the ***British Journal of Ophthalmology***. 1931. Price, 20s.

If it be true of medical literature that its amount is in direct proportion to the obscurity of the subject with which it deals, it is not surprising to realize the output which has accumulated in reference to detachment of the retina, particularly when we reflect that the condition is one of which the pathology is variable and in most instances still remains obscure. The author, with painstaking care, has gathered all that is known and every theory which has been advanced in regard to detachment of the retina, and in this volume includes a bibliography which cannot fail to be of assistance to those who will in the future work on the pathological questions of which so few are as yet settled.

In discussing aetiology, Anderson regards all cases of retinal detachment as secondary, but states that in every case it is necessary to apprehend the condition to which it is secondary, and that no diagnosis is complete unless this can be ascertained: as far as the vitreous humour is concerned the slit-lamp promises to be of some assistance.

Chapter II is devoted to the structure and functions of the tissues involved, emphasizes many important developmental and anatomical facts upon which little stress is usually laid, and contains many shrewd and suggestive observations by the author.

The bio-physical views of Duke-Elder are concisely stated, and the hope entertained that further work in this and in the bio-chemical direction will be useful.

Chapter III on pathogenesis is comprehensive; every theory is reviewed and in the last five pages of this chapter the author usefully summarizes the conclusions which he has drawn.

In the chapter on differential diagnosis the methods and value of transillumination are very fully explained.

In dealing with treatment the new and promising methods of ignipuncture necessarily receive full consideration, but the older therapeutic methods, operative and otherwise, are not neglected.

The book is produced in a style well worthy of the immense and valuable labour which the author has bestowed upon it, and should be read by every surgeon: it is an excellent monograph, produced at the opportune moment, and will add to the reputation of British ophthalmology.

Studies in the Photo-Activity and Therapy of the Tungsten-Titanium Arc. By J. BURDON-COOPER, M.D., B.S., B.Sc. (Durh.), F.R.C.S.E., D.O. (Oxon.), F.C.S., and ARTHUR ROBERTS, T.D., F.R.C.S.E., M.R.C.S.Eng. John Wright & Sons, Ltd., Bristol, and Simpkin, Marshall, Ltd., London. 1931. Price, 10s.

So little is really known about the properties and action of ultra-violet light, that a book of this nature is very welcome. The authors have expended a great amount of time and trouble in contriving experiments to investigate this question. These experiments, and the various apparatus actually used in treatment, are illustrated by numerous excellent photographs.

It is made quite clear that the best results are obtained from an electrode composed of 98 per cent. tungsten, 1·5 to 2 per cent. titanium, with a trace of chromium, and that this superiority is in part due to the steady emission of the late red and infra-red rays, which increase the permeability of the body tissues, and help to improve vitality and resistance to infection.

With regard to the penetrative power of ultra-violet light, it used to be held that this was limited to 9·5 mm. at most, but, using the tungsten-titanium arc, the rays pass through 5/16 in. of recent

ox-pelt with its subcutaneous tissues, and even through the thickness of the terminal phalanx of the third finger. It is the addition of the small amount of titanium to the electrode which increases the penetrative power of the therapeutic ultra-violet light rays, and gives a spectrum of more even intensity than that obtained from either tungsten or titanium alone. Various materials have been examined in respect of their power of stopping the bactericidal rays of ultra-violet light, and it is shown that Chance's glass offers least resistance, while quartz, ox-pelt, "windolite," "vita glass," cardboard, and ordinary glass present increasing obstruction.

Though, in moderation, the red and infra-red rays assist penetration, yet too much of this type of radiation causes heating in excess of the body temperature, and the penetration is diminished. There is an optimum temperature of the tissues at which penetration, bactericidal power, and cutaneous chemical changes are at their highest.

Analysis of an arc shows that the longer wave lengths give rise to molecular alteration and thermal changes, while the shorter produce molecular disintegration, photo-abiotic, chemical and electrical changes. The shorter waves of ultra-violet light are largely absorbed by the surface layers of the skin, while the longer ultra-violet rays reach the subepidermal tissues, and visible rays attain a greater depth.

A chapter of illustrative cases is included which shows the results obtained in various diseases.

This book, clearly and simply written, will appeal mainly to actinotherapeutists, who will find in it a useful and interesting store of information.

NOTES

Deaths

We regret to have to record the recent death of Dr. Julian Augustus Lea, who practised for many years in Grahamstown, South Africa. Dr. Lea was one of our South African representatives on the General Editorial Committee of this Journal and contributed three papers to our fourth volume. He qualified at the "Colleges" as far back as 1874, from Charing Cross Hospital; took the M.B. Toronto in 1882, and the F.R.C.S.Edin. in 1893, and became a member of the Ophthalmological Society of the United Kingdom in 1895. He retired 10 years ago and resided in Natal. Mr. Seale, of Grahamstown, the author of a paper in our September number (p. 514) has been so good as to send us a cutting from *The Farmer*, of Maritzburg. It appears that Dr. Lea, after his retirement from active ophthalmic practice, took an interest in farming, and the

health of his neighbours in general. He had been actively engaged in medical work during the influenza epidemic which ravaged Natal in June last, and succumbed to the disease himself.

The death is also announced of Dr. L. Webster Fox, of Philadelphia, aged 78 years.

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Appointment

MR. L. H. SAVIN has been appointed Junior Ophthalmic Surgeon to King's College Hospital.

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Corrigendum

MR. HARRISON BUTLER calls attention to the fact that, by an oversight in proof correction, the average initial convergence in a series of cases of squint submitted to operation is given on page 492 as 14. The figure should have been 34.

FUTURE ARRANGEMENTS

1931

November 6.—North of England Ophthalmological Society, at Bradford.

November 13.—Section of Ophthalmology. Royal Society of Medicine.

December 1.—Midland Ophthalmological Society, at Birmingham.

December 4.—North of England Ophthalmological Society, at Leeds.

December 11.—Section of Ophthalmology, Royal Society of Medicine. (Clinical Meeting).

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January 8.—Section of Ophthalmology, Royal Society of Medicine.

February 5.—North of England Ophthalmological Society, at Newcastle-on-Tyne.

February 12.—Section of Ophthalmology, Royal Society of Medicine.

March 4.—North of England Ophthalmological Society, at Liverpool.

March 11.—Section of Ophthalmology, Royal Society of Medicine. (Clinical Meeting).

April 8.—North of England Ophthalmological Society, at Sheffield.

June 10.—Section of Ophthalmology, Royal Society of Medicine. (Annual Meeting).

CONTEMPORARY OPHTHALMIC LITERATURE

American Journal of Ophthalmology. *September, 1931.*

FUCHS. Contribution to the anatomy of congenital ectropium uveae (flocculi iridis).
LEMON. An internist's inquiry regarding the etiologic factors of uveitis.
FRY. The pathology of papilloedema.
BLACKMAR. Xeroderma pigmentosum.
HILGARTNER and HILGARTNER, JR. Deep orbital injury by stick of wood; exophthalmos and optic atrophy following.
MARCOVE. Parathyroid cataract.
WENTWORTH. Variations of the normal blind spot with special reference to the formation of a diagnostic scale.
HOLZER. Ophthalmology in aviation medicine.
MAYER. Epinephrin in progressive myopia.
TOOKER. Annular scleritis.
HUGHES. An improved double rotary prism.
MILLS. The possible relation of strong lights in the motion picture industry to lens changes.
BOTHMAN. The relation of the basal metabolic rate to progressive axial myopia.
MCALESTER, JR., and MCALESTER, THIRD. Nickel sensitization from white gold spectacle frames.
EMMONS. Ocular symptomatology in acute poliomyelitis.
GRESSER. Ocular disease and the vermiform appendix.
SCARLETT. Senile macular changes of the retina associated with focal infection.
GRADLE. Concerning simple glaucoma.
GOLDENBURG. A glaucoma study.
JENNINGS. Evaluation of pseudo-isochromatic tests for the detection of colour blindness.

Annales d'Oculistique. *August, 1931.*

NICLOUX and REDSLOB. The passage of ingested alcohol into the vitreous and the lens.
TERSON. Posterior cautery puncture in absolute glaucoma after a retinal detachment.
ROUSSEAU. Hypertonus in acute iridocyclitis. Indications for surgical treatment.
COSMETTATOS. Epithelial plaques of the cornea.
ARNOULD. Antigen therapy in ocular tuberculosis.
BETTREMIEUX The curability of detachment of the retina.
PAVIA. The photo-topographic shifting of the retina when the detachment is behind the equator.
ROURE. Concerning the replacement of corneal incisions recently proposed for cataract operation.
DE SOUSA. A new method of ocular surgery. Use of ultra-violet light.
TERSON. Akinesia of the superior rectus and the orbicularis by the conjunctival route.
——— A better arrangement of electric lighting of the operative field. (Auto-photophore with cervico-thoracic attachment).
MAUGER. A lever lid retractor.
DE LEMOS. A case of exophthalmos of unknown cause.

Archives d'Ophtalmologie. *September, 1931.*

WEEKERS. The usual laws of experimental ophthalmotonic reactions, direct and consensual, produced by different procedures.
TERRIEN and COUSIN. Epithelioma of the limbus.
MARIN-AMAT. A late and serious complication of dacryo-cysto-rhinostomy.

Archiv für Ophthalmologie. *September, 1931.*

KOYANAGI. The question of the pathogenesis of pigmentary degeneration of the retina.
V. HIPPEL. Angiomatosis retinae and Coats' retinitis exudativa. Pseudoglioma from tuberculosis
——— On angioma of the choroid.
KRAUER. Chronic eye changes in cattle.
KRAVKOV. The difference threshold of the periphery of the retina in dark adaptation.
DYMSCHITZ. A case of unusually well-developed iris flocculi.
WAGNER. The determination of the linear measurements on the superficial surface of the eye from the limbus to the ora serrata, to the posterior pole, and to the optic disc.
BLATT. Experiences with cautery puncture.
MENNINGER-LERCHENTHAL. Congenital anophthalmos.

Bollettino d'Oculistica. *July, 1931.*

DI MARZIO and SABBADINI. On the treatment of detached retina by Gonin's operation.
ANTONIBON. Graphic representation of ocular movements in writing.
BARLETTA. On a case of cysticercus cellulosae.
BIOZZI and FAVIA. Ocular diphtheria in the newborn contracted during labour.

Lettura Oftalmologica. *July, 1931.*

CASO. Sclerotic cartilage in the vertebrates.
DOMINEDO. Orthophoric correction in ametropia.
MONTANELLI. Subconjunctival cyst from Coenurus cerebralis.

Revista de Ophthalmologia. *August, 1931.*

PAVIA. Alterations of the reflexes from the limitans interna as an early sign of hardly visible lesions.
VAMPRE. Traumatic hemianopia.
DE ALMEIDA. Artificial maturation of cataract.
MATTOS. Traumatic scleral cysts.

Archivos de Oftalmologia Hispano-Americanos.

September, 1931.

CARRARAS. Iridectomy *ab externo*.
LUQUE. Solitary retinal hypertension of vascular origin.
AMAT. Treatment of conjunctivitis in general.
CORO. Tuberculous keratitis.

Boletin de Informacion Oftalmologica. *July-August, 1931.*

DELLEPIANE. On perforating wounds of the cornea.
TRIVISONNO. A case of sympathetic ophthalmitis.
ONETO. Ocular complications in a case of Werlhof's disease. Essential thrombopenia.

Revista Oto-Neuro-Oftalmologica y de Cirugia Neurologica.

August, 1931.

MONIZ, PINEO and LIMA. Some cases of cerebral tumours rendered visible by encephalography.
PAVIA. On two cases of tumours of the hypophysis.